ASTHMA

KINGSCOTE

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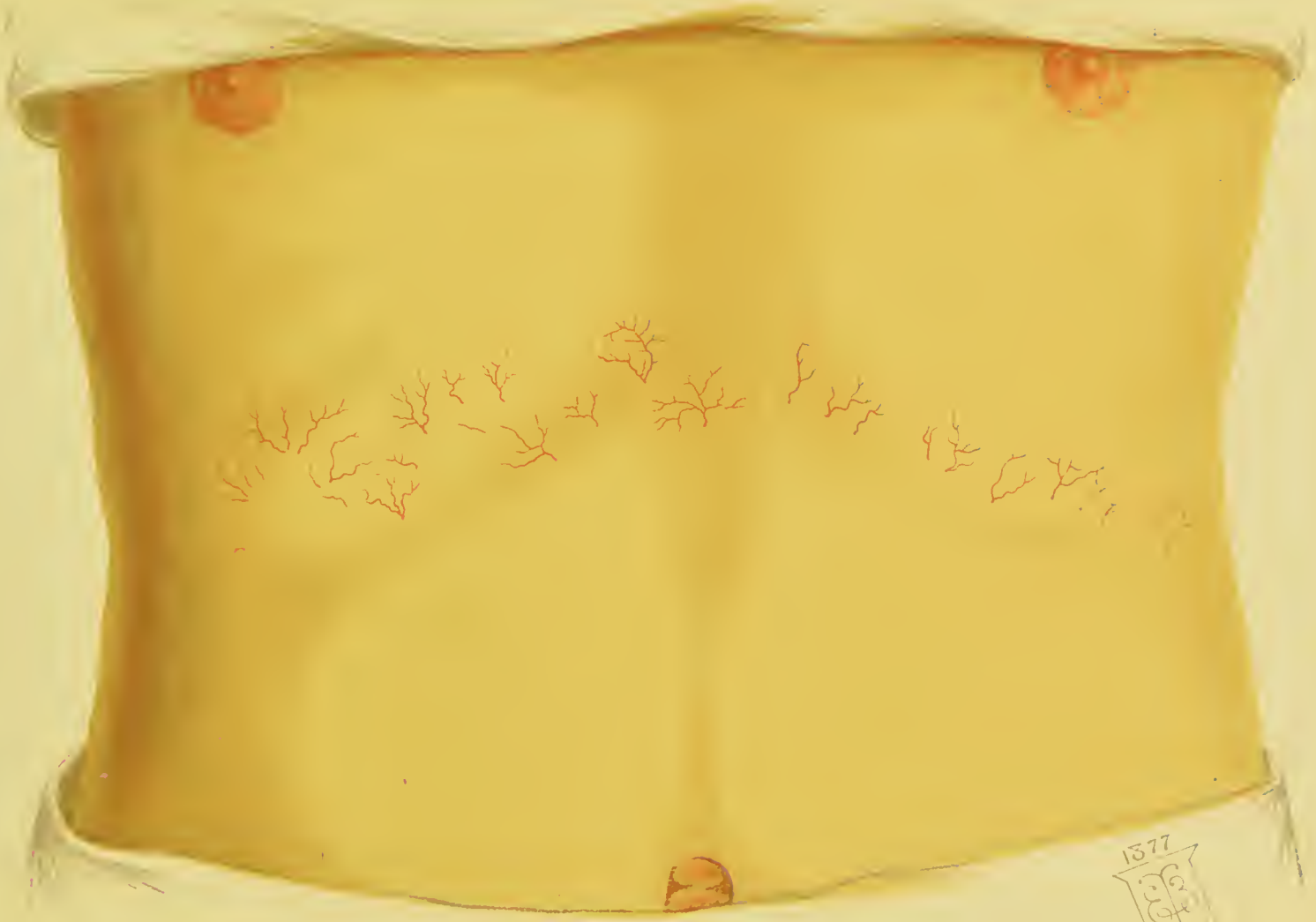

The CINGULA ATHLETICA (Blake).

A pathognomonic sign of Hypertrophic Emphysema in the gouty.

ASTHMA:

RECENT DEVELOPMENTS IN ITS TREATMENT.

BY

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WITH COLOURED FRONTISPIECE AND ILLUSTRATIONS.

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PREFACE.

THIS book makes no pretension to be considered an exhaustive treatise on Asthma. It merely consists of the record of a number of observations taken during some years' special practice in that subject. As will be seen, the earlier results were obtained empirically; and some theories had to be woven to account for the phenomena. We discover empirically only such secrets as Nature chooses to divulge, and it is for us to make them fit in with her known laws—*exempli gratia*, Sir Isaac Newton's apple and James Watt's kettle.

It chanced that, while working at the treatment of chronic heart disease, I was able to relieve some patients of their Asthma (see page 103), and I now suggest some possible reasons for the relief obtained. In order to fully comprehend the theories alluded to, it is necessary to dip somewhat deeply into the consideration of certain physical facts.

As the diagnosis depends very much on percussion, which latter, by the ordinary methods, is difficult of achievement in Asthma, owing to the extreme frequency of Emphysema in these cases, I have thought it advisable to devote a Chapter to the consideration of Percussion

and to a description of a new pleximeter, which overcomes the difficulty alluded to. Without this pleximeter, I should have been unable to make the accurate diagnoses which have led to the evolution of the theories hereafter suggested. These, although doubtless containing some fallacies, possess, I fully believe, a germ of truth.

The busy practitioner, therefore, who may be unable to devote the time to these considerations, will do well to pass at once to Chapter XII, where the study of Asthma proper begins.

My thanks are due to the many friends who have given me so much help in compiling this little work, more especially to Professor Keiller of Texas, Dr. Gibson of Edinburgh and to Dr. Harry Campbell of London; who have so kindly permitted me to quote largely from their writings. To Drs. Maguire, Harry Campbell and to Mr. Barnard, who have helped me to revise the proofs, also to Professor Leonard Hill, to whom I am indebted for most valuable information with regard to the pericardium. Last, but not least, my grateful thanks are due to my former chief at the Royal Infirmary of Glasgow, William Macewan, now Professor of Clinical Surgery at the Western Infirmary, whose brilliant example first incited me to habits of observation.

ERNEST KINGSCOTE.

31, Lower Seymour Street,
Portman Square, W.
May, 1899.

LIST OF ILLUSTRATIONS.

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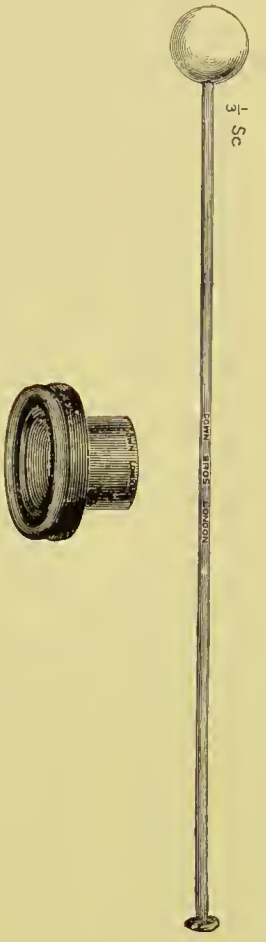


KINGSCOTE'S PLEXIMETER.

ASTHMA;

RECENT DEVELOPMENTS IN ITS TREATMENT.

CHAPTER I.

PERCUSSION.

Ever since 1761, when percussion as a means of physical diagnosis was first suggested by Avenbrugger of Vienna, it has been generally recognised that deep percussion of the thorax is not free from uncertainties arising (1) from the vibrations of the chest-wall veiling the note really given out by the structure underlying the plessor, and (2) from the fact that percussion, when sufficiently hard to produce a true note, is apt to hurt or at least alarm the patient, and (3) from the fact that the organ to be percussed out sometimes underlies an air-containing viscus. I have devised a pleximeter which overcomes all these difficulties. It consists of a felt cone made with a shoulder to which a thick indiarubber ring is attached in such a manner that, when the instrument is firmly pressed against the chest, the apex of the cone touches a definite point on the skin in the centre of the ring, which ring cuts off the extraneous chest vibrations, so that if the other end of the cone be now

struck smartly with a hammer having a flexible handle, the true note of the underlying structure is elicited.

My results have been verified experimentally on recently killed sheep, pigeon-breasted animals, which are by no means easy subjects for percussion. *The lungs not being artificially inflated*, I percussed the heart-boundaries in the usual manner, using the finger as a plessor and marking out the boundaries in ink. Long skewers following the ink lines were then thrust right through the thorax, and on opening the chest they were found to be surrounding and touching the pericardium. On this experiment being repeated with my pleximeter a similar result was obtained. Another similar experiment was then made, the difference being that *the lungs were artificially inflated*, when it was found that the skewers pushed through the boundaries marked by the ordinary method of percussion penetrated the heart muscle one inch from the ultimate margin on the left side, and a quarter of an inch on the right side of the heart; but with the boundaries marked by my pleximeter, the skewers pierced the overlapping lung substance for a distance of about one and a half inches on the left side, and half an inch on the right side, and were found just grazing the actual cardiac boundaries. In this case even the kidneys were percussed out with the pleximeter from the back and the skewers again showed that the percussion had been accurate. It was thus proved

conclusively that the anatomical dimensions of the heart had been correctly percussed out, even although overlapped by a considerable amount of lung substance. A fresh sheep's heart was then suspended by means of a string in a cardboard box, eighteen inches long and eight inches deep, such as is used by milliners, the direction of the cardiac diameters being unknown to the percussor. The least distance of the heart's surface from the lid of the box was about an eighth of an inch. By the ordinary method it was impossible to percuss out the heart boundaries, but by using the pleximeter their positions were exactly defined. In this case the heart was of course entirely surrounded by air, and the ordinary percussion method only gave a duller note when that position was reached where the heart almost touched the box, and corresponding to the triangle of supra-cardiac dulness of the text books.

A ready way of demonstrating the advantages of the pleximeter is to carefully percuss out the boundaries of the heart on a normal chest with the ordinary method, and mark the result on the skin, then to repeat the performance with the pleximeter and the two results will tally. Now direct the patient to take a very deep breath and hold it (thus covering the heart with air-distended lung and simulating emphysema); the line of left heart dulness will be found to have receded from just within the left mid-clavicular line to about the sternum. If the experiment

be repeated with the pleximeter, the line of left heart dulness will be found in its normal position, because the peculiar attributes of the pleximeter admit of accurate percussion through an air-distended viscus.

The fact that all these experiments have been repeated many times with invariably uniform results, proves the possibility of obtaining an accurate definition of the boundaries of the heart, even when it is overlapped by lung tissue. A special form of hammer is used with the pleximeter, its head being made of lamb's wool so compressed that it cannot be beaten out of shape, a necessary condition for the production of an absolutely true note, and it is of such a weight that the required note is obtained without the absorptive power of the surrounding ring being overcome. The finger may, however, be employed as a hammer with equally good results when the wrist is very flexible. In using the pleximeter it must be applied firmly to the region to be percussed, and there must be no alteration in its position when it is struck with the hammer. The instrument has the further advantage that the sense of vibration is retained whilst using it. The manufacturers are Messrs. Down Brothers, St. Thomas Street, Borough.

Trousseau, when lecturing at the Hotel Dieu, Paris, on percussion, said: "The ideal of percussion is to percuss the smallest possible surface so as to disturb the smallest possible portion of an organ, to percuss in such a way that

at a distance of a few millimeters the space not percussed should not be made to vibrate, and should not mingle its sounds with the sounds of the percussed space." An ideal which this plexímeter completely realises.

This was emphasized by the late Dr. August Schott, of Nauheim, in the *Centralblatt fur Medecinischen Waarschaften*, some years ago, and he devised a plexímeter which in some measure overcame the difficulties in question.

This chapter has a special bearing on Asthma from the fact that it suggests a method of accurately percussing the heart margin, although overlapped by emphysematous lung.

Radiography as a means of determining the cardiac diameters will probably be a useful method in the future the subject is being well worked out by Sequeira, but some of the skiagraphs I have seen have not been very satisfactory.

Owing to the heart being in constant movement, I doubt whether we shall get the definite information we require until we have learnt to take snapshots through a Crookes' tube. Besides, we do not quite know the effect of the Röntgen Rays on the organisms of the body, and several people have told me that they have had very peculiar feelings while under their influence. Cases have been recorded of internal abscess supposed to have been due to the effect of the rays. In addition, although the methods

might be very useful to settle some disputed point of diagnosis, they would be inconvenient for application in daily practice. Doubtless, however, the genius of the future will solve this difficulty; should the rays be proved to be harmful, some means will be discovered to obviate their deleterious effects.

Much of this chapter has already been published in the *Lancet* for December 19th, 1896.

CHAPTER II.

APPROXIMATE ANATOMY OF THE LIVING HEART.

Before proceeding to the study of cardiac dilatation, it is advisable to consider the normal heart as it exists in the living body, because the heart which we see in the post-mortem room gives us a very poor idea indeed of that organ as it exists during life. I cannot do better than quote from an article on the "Descriptive Anatomy of the Human Heart" by William Keiller, F.R.C.S.E. (Professor of Anatomy, University of Texas, published in the *American Journal of Medical Sciences* for April, 1898). Prof. Leonard Hill kindly undertook to test the accuracy of the diagrams for me. He writes as follows from the physiological laboratory of the London Hospital:—

"The Heart drawings are like our formaline injected bodies and are much more accurate than ordinary text-book plates."

The magnificent works of Braune and His have, within the past decade, revolutionized many sections of descriptive and topographical human anatomy. Till lately, dissection of the fresh or imperfectly prepared subject was

the only means used to arrive at a knowledge of the form and relations of each structure; but within recent years, the examination of the body made while frozen, and the dissection of subjects hardened to comparative rigidity by continued intra-arterial injection of such hardening agents as chromic acid, chloride of zinc or corrosive sublimate, have corrected grave errors which had arisen from the exclusive study of the organs in the flaccid condition met with when ordinary methods are used. Even on the post-mortem table, and within thirty-six hours of death, the liver, spleen, and heart "flop" out of all shape (I can think of no more expressive word, if it be somewhat inelegant), and are no more like the same organs as seen when hardened *in situ*, than the jelly-fish, half embedded in the sand—a shapeless and repulsive mass—resembles the living embodiment of symmetry and beauty propelling itself through the clear blue water. What, then, must be the condition of the same organs weeks or months after death, in bodies preserved by freezing or by arsenical solutions which do not harden the tissues? Verily, such organs are the despair of the medical student; and those who have never seen hardened organs may well be excused should they regard the models of His, and allied descriptions, with a certain amount of respectful incredulity."

Sueh, I eonfess, was my own feeling toward them till, on the adoption of my present method of preserving bodies, I found to my surprise that these and other organs were thereby sufficiently hardened to present almost perfeet eounterparts of these models, without any interference with the methods of dissection. Thus there is now in my dissecting-room scarcely a liver, kidney or spleen removed by the student from his subjeet, that does not exhibit in perfeetion all the surfaces, borders, and impressions, described and modelled by His.

While the text-book descriptions of liver, kidney and spleen have been changed to accord with recent views, it is somewhat strange that the heart is still portrayed as it used to be many years ago; and that though it requires the most vivid imagination and elastie eonscienee to reeoncile the description with the accepted model of His. And this is still more strange, since elinieians have found the text-books' description of the organ so utterly inadequate that they have had to invent terms of their own in order to indicate anatomical facts in the living—terms which have no place in the dissecting-room vocabulary. I have long felt that though the description of the heart as presenting a base, apex, right and left borders, and anterior and posterior surfaces, is sufficient to describe the flabby, shapeless mass one finds on the post-mortem table when

the excised organ lies before one, yet it is useless as applied to His's model or to my own specimens hardened *in situ*. I have, therefore, for the past few years described in my lectures what I saw before me, without reference to the text-book; and I feel that the matter is of sufficient importance to merit presentation to my brother anatomists.

When I consider that my own description is immensely more complex than that now in vogue, I feel some little compunction in suggesting an additional burden to the already sorely laden student of anatomy; but I am encouraged by the remembrance of what has happened in the case of the liver. That organ which, in the primitive simplicity of old text-book descriptions, had only two borders, two surfaces, and no impressions at all, has now five borders, as many surfaces, and eight impressions; but everything is so definite, its relations are so evident, and the picture can be so vividly printed on the brain, that what was before a pure matter of memory and a puzzle to the student, is now easily described and readily understood and remembered.

First let me allude to the inconsistencies in our accepted descriptions. The pericardium is spoken of as cone-shaped, its apex upward, its base resting on the diaphragm. Yet the heart which, with the commencement of the great

vessels completely fills it, is described as a cone with its apex directed downward, forward, and to the left, and its base upward, backward and to the right. But this is a small matter, and probably justifiable; graver inconsistencies are to come.

Clinical manuals, such as Gibson and Russell's *Physical Diagnosis*, and articles on topographical anatomy, such as that in Morris's *Treatise*, discard the word "base," or explain that in their use of it they mean the "upper limit": use "lower border" to signify what in the dissecting-room we call the "left border," apply the term "right border" to what in the dissecting-room we do not describe at all; and their "left border" is anything but synonymous with the left border of the descriptive anatomist.

Having, I trust, succeeded in showing that some change is necessary, I shall endeavour to submit to you a description of the external configuration and relations of the human heart which shall be accurate, concise and free from redundancy. I would beg that those who honour my description when in print with a careful perusal, will compare it and the illustrations with the model by His; and those who desire to verify it in the dissecting-room may do so, in any body injected through the right common carotid or femoral artery with a gelatine injecting mass. (My description is beautifully illustrated by specimens

obtained from bodies prepared by injection of formaldehyde 5 per cent. of a 40 per cent. solution.) The drawings are made from a rather enlarged specimen, of which the right side was filled with coagulated blood and the left side with injection mass; but, though large, it agrees in all essential matters with His's model and many other dissecting-room specimens which I have carefully examined. My description is markedly borne out by vertical, sagittal, mesial and vertical-coronal sections of the frozen body; a horizontal section I have not yet been able to compare with it.

Thus viewed, the heart is an irregular, four-sided pyramid, whose base rests on the diaphragm, and whose apex has been, as it were, removed to afford attachment for the ascending trunks of the great vessels. It thus presents for examination five surfaces (including the base), borders separating these, an anatomical apex, and a "clinical apex," a term which I feel compelled to retain because it is almost inseparable from physiological and clinical phraseology.

The anterior surface (Fig. 1), the first which meets the eye when the chest and pericardium are opened, is triangular in shape, slightly convex, and is directed forward and a little upward, being in sagittal mesial section parallel with the sternum. It includes the greater part of the right

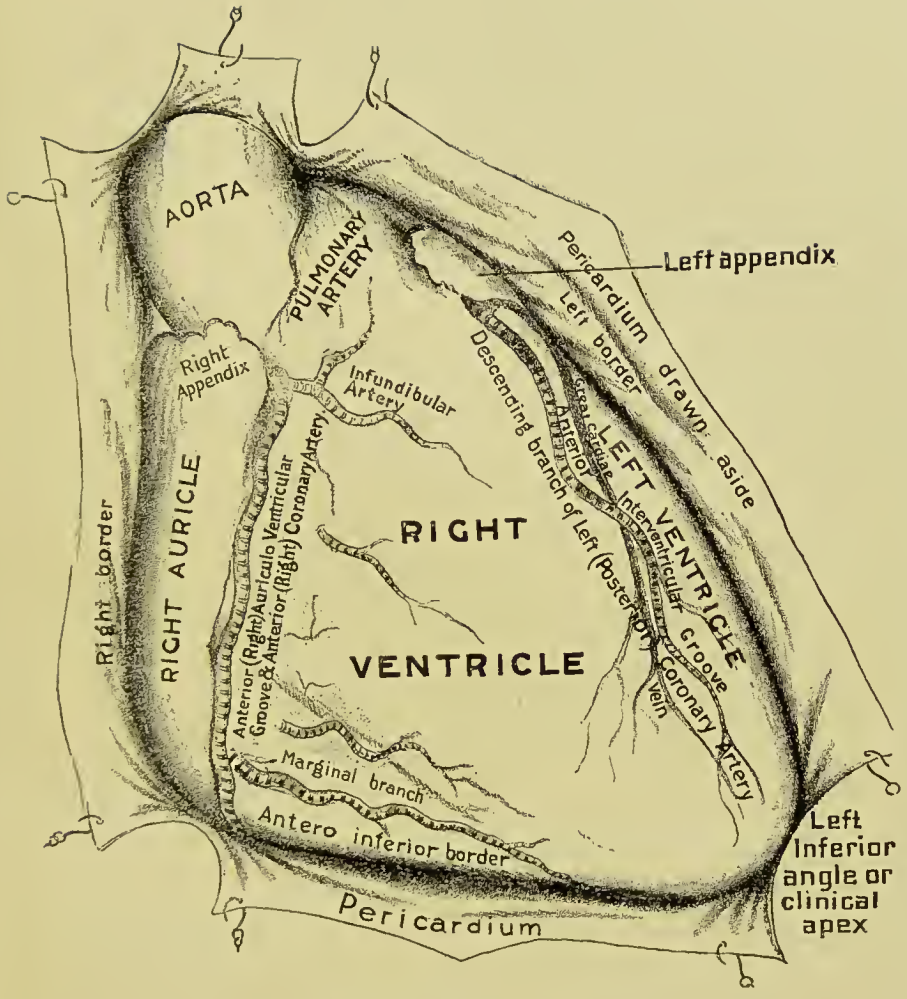

AORTA
PULMONARY ARTERY
Pericardium drawn aside
Left appendix
Left border
Right Appendix
Infundibular Artery
Descending branch of Left (Posterior) Coronary Artery
LEFT VENTRICLE
Anterior Interventricular Groove
Right border
RIGHT AURICLE
Anterior (Right) Auriculo Ventricular Groove & Anterior (Right) Coronary Artery
RIGHT VENTRICLE
Coronary Vein
Coronary Artery
Marginal branch
Antero inferior border
Left Inferior angle or clinical apex
Pericardium

FIG. 1.

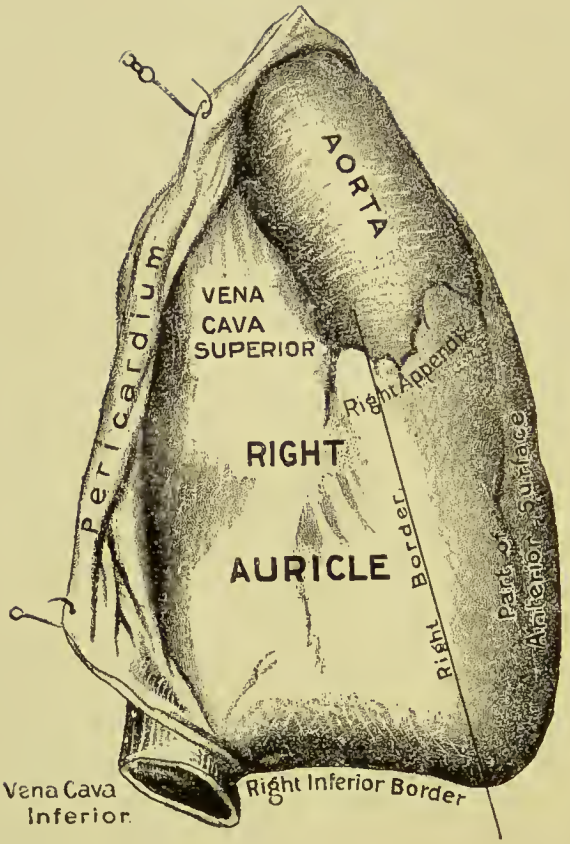

AORTA
VENA
CAVA
SUPERIOR
Pericardium
Right Appendix
RIGHT
AURICLE
Right Border
Part of Anterior Surface
Vena Cava
Inferior
Right Inferior Border

FIG. 2.

ventriele, and portions of the left ventriele, left aurieular appendix, the whole right appendix, and part of the right auricle. It is bounded below by the sharp, almost straight antero-inferior border (margo aeutus), on the left by the oblique, eonvex, and slightly rounded left anterior border (the left border of clinieians), and on the right by the nearly vertieal and convex right anterior border (the "right border" of elinieians). Its superior angle marks the anatomical apex, and here the surface merges into the anterior walls of the aorta and pulmonary artery. Its left anterior angle forms the clinical apex.

On this surface are seen the anterior or right coronary artery in the anterior aurieulo-ventrieular groove, while its marginal braneh runs along the antero-inferior border; and in the anterior interventrieular groove is the descending branch of the posterior or left eoronary artery, aecompanied by the great eardiac vein.

Relations.—Separated from it by the pericardium, are the margins of the lungs and pluræ, the sterno-perieardial ligaments, triangular sterni, internal mammary vessels, and sternum.

The right surface (Fig. 2), is markedly convex, four-sided, lies almost vertical, and is directed toward the right. It includes the greater part of the right auricle. Its anterior, posterior and inferior borders are only slightly

rounded, and are therefore fairly well defined. At its superior extremity the surface blends with the wall of the superior vena cava, and at its posterior inferior angle it is similarly related to the vena cava inferior.

Relations.—It is separated by pericardium from the right phrenic nerve and vessels, pleura and inner surface of the right lung.

The left surface (Fig. 3), is a convex, triangular area, directed mainly upward and toward the left. It includes about one-half of the free surface of the left ventricle and the left auricular appendix. It is separated from the anterior surface by the left border; from the inferior surface by a rather sharp left inferior border; and behind it is bounded by the left pulmonary veins and left auriculo-ventricular groove with the coronary vein embedded therein. It presents the proximal extremities of the descending branch of the left coronary artery and great cardiac vein, the marginal and transverse branches of the same artery, and the posterior cardiac and coronary veins.

Relations.—It is separated by pericardium from the left phrenic nerve and vessels, left pleura, and inner surface of the left lung.

The posterior surface (Fig. 4) (dorsal surface) is called "the base" in text-book descriptions. It is four-sided, rather

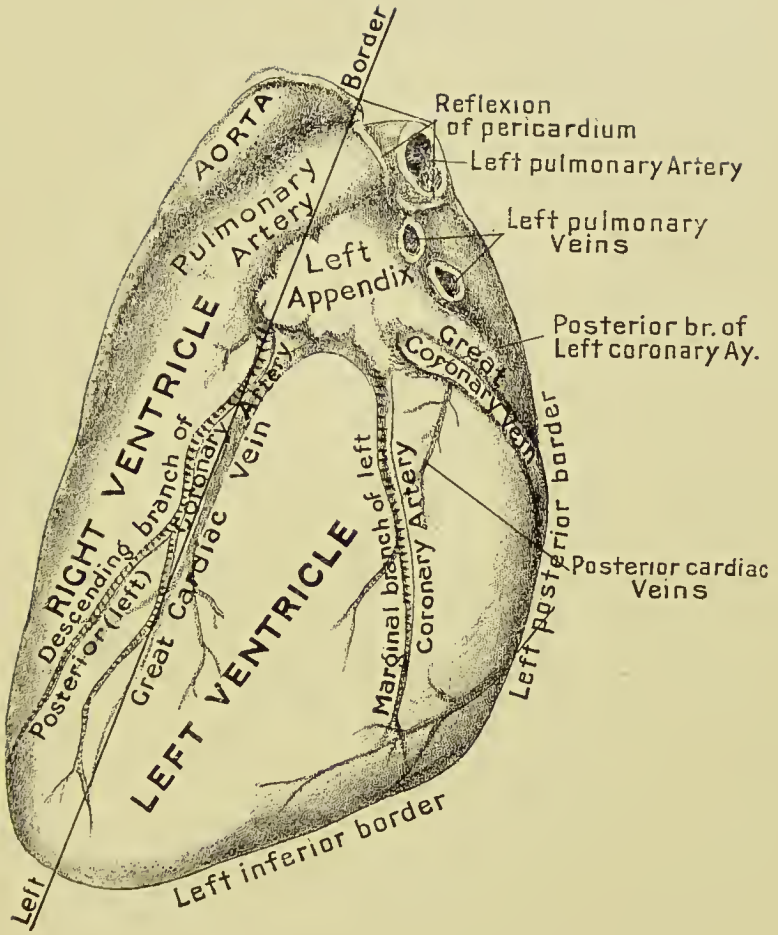

Right Border
Reflexion of pericardium
Left pulmonary Artery
Left pulmonary Veins
AORTA
Pulmonary Artery
Left Appendix
Posterior br. of Left coronary Ay.
Great Coronary Vein
RIGHT VENTRICLE
Descending branch of Coronary Artery
Posterior (left) Great Cardiac Vein
LEFT VENTRICLE
Marginal branch of left Coronary Artery
Left posterior border
Posterior cardiac Veins
Left
Left inferior border

Fig. 3.

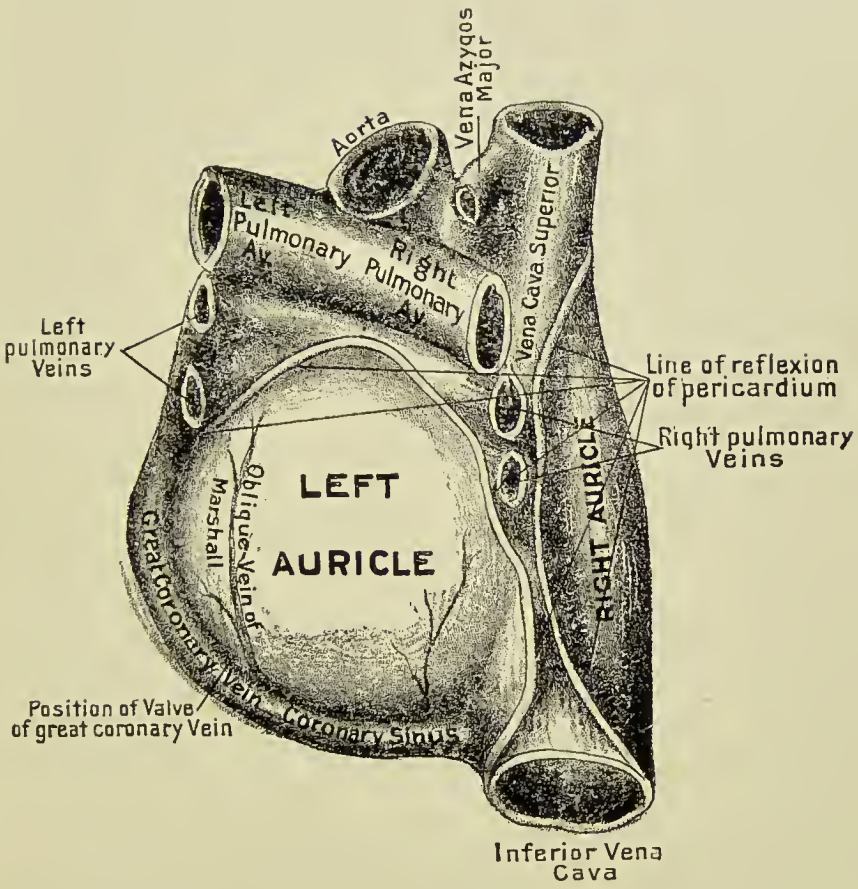

Vena Azygos Major
Aorta
Left Pulmonary Ay.
Right Pulmonary Ay.
Vena Cava Superior
Left pulmonary Veins
Line of reflexion of pericardium
Right pulmonary Veins
LEFT AURICLE
RIGHT AURICLE
Oblique Vein of Marshall
Great Coronary Vein
Position of Valve of great coronary Vein
Coronary Sinus
Inferior Vena Cava

Fig. 4.

narrower above than below, is convex, vertical, and directed backward. It is formed by the left auricle and by the portion of the right auricle which joins the two venæ cavæ behind, and is bounded by rather sharp and well-defined borders. It is bounded below by the inferior vena cava and the coronary sinus, above by the right pulmonary artery, on the right by a fairly defined border joining the two venæ cavæ, and on the left by the cardiac openings of the left pulmonary veins and by the great coronary vein. It presents the openings of the coronary veins (right and left), the great coronary vein and coronary sinus, and the oblique vein of Marshall, which last runs downward over the surface, to enter the left extremity of the coronary sinus. It is only partially invested by the serous layer of the pericardium.

Relations.—It is separated by pericardium from the bronchi, œsophagus *and vagi*, descending aorta, vena azygos major, and thoracic duct.

The inferior surface (Fig. 5) (diaphragmatic surface or *base*) is quadrilateral, slightly convex or almost flat when the ventricles contain blood, slightly concave when they are empty and relaxed. It is directed downward and a little backward and toward the right, and is bounded by rather sharp and well-defined borders. It is formed by a small portion of the right auricle and opening of the vena

cava inferior, the rest of the surface being about equally divided between the right and left ventricles. Behind the vena cava is seen a small portion of the left auricle. In addition to the inferior caval opening, it presents the inferior extremities of the right (anterior) and left (posterior) interventricular grooves, with the right coronary artery embedded in the former and the coronary sinus in the latter. Crossing it diagonally is the inferior interventricular groove, with the descending branch of the anterior (or right) coronary artery and middle cardiac vein. The posterior cardiac vein runs along its posterior border.

Relations.—It is separated by the central tendon of the diaphragm, and some diaphragm muscle from the superior surface (impressio cardiaca) of the liver.

The *apex* of the pyramid is formed by the aorta (Fig. 1); pulmonary artery (Fig. 3) and superior vena cava (Fig 2); these structures spring from the heart on a level with the upper margin of the third costo-sternal articulation, extending an inch and a half to the left, and one inch to the right of the middle line. This we may conveniently name with clinicians the upper limit of the heart.

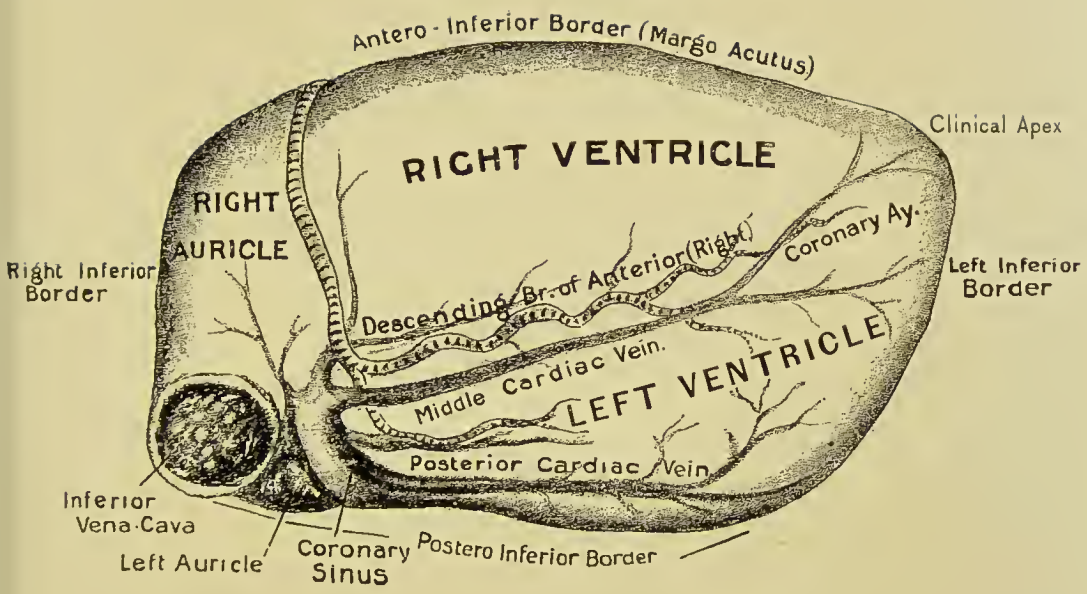

Antero - Inferior Border (Margo Acutus)
Clinical Apex
RIGHT VENTRICLE
RIGHT AURICLE
Right Inferior Border
Descending Br. of Anterior (Right)
Coronary Ay.
Left Inferior Border
Middle Cardiac Vein.
LEFT VENTRICLE
Posterior Cardiac Vein
Inferior Vena Cava
Left Auricle
Coronary Sinus
Postero Inferior Border

FIG. 5.

Superficial Indications.

The *upper limit* of the heart has just been indicated. The left half of this line will mark the position of the pulmonary and aortic valves.

The *clinical apex* is indicated by a point between the fifth and sixth ribs, three and one-half inches to the left of the middle line.

The *antero-inferior border* is to be indicated on the chest wall by an oblique line, slightly convex downwards, extending from the clinical apex on the left across and slightly upward, to a point one inch to the right of the middle line at the level of the sixth chondro-sternal articulation. Along this line the cardiac blends insensibly with hepatic dulness.

These lines being drawn, the right and left borders of the heart's anterior surface or the absolute lateral limits of the heart's dulness will be defined by convex lines joining respectively their right and left extremities. Thus, on a level with the fourth chondro-sternal articulation, the area of the heart's dulness extends three inches to the left, and one and three-quarter inches to the right of the middle line.

It would seem more consistent with the above description to change some other elements in the cardiac nomenclature; for instance, the interventricular grooves are seen

to be superior and inferior; the *right coronary* artery would then be named *anterior*, and its branches respectively infundibular (as at present), right ventricular (now "marginal"), and inferior interventricular (now descending); the left coronary artery would be better named posterior, and its branches superior interventricular (now "descending"), left ventricular (now marginal), and auriculo-ventricular (now "transverse").

CHAPTER III.

CARDIAC DILATATION.

Referred to by the older anatomists, dilatation of the heart was more particularly noticed by Harvey and Willis. Mayow described dilatation of the left auricle and right ventricle as a result of mitral obstruction, and Vieussens mentioned an instance of dilatation of the left ventricle resulting from aortic regurgitation. The method of production was carefully analysed by Senac, and the effects produced on the circulation were detailed by Morgagni. All the great writers upon diseases of the heart, from his time downwards, have dealt fully with the condition.

The first recognition of the clinical features of cardiac dilatation is really due to Corvisart, who observed the increased area of pulsation. The means of recognition by auscultation was carefully studied by Laennec (see p. 74). Bertin, in addition to extending previous observations upon the symptoms of dilatation, distinguished between different varieties, and his classification has been very generally adopted even to the present time. (Gibson).

Dilatation of the heart is much more frequent than is generally supposed; it frequently remains undiagnosed because, (*a*) it is often not looked for, and, (*b*) in many cases the ordinary methods of percussion are inadequate. (See Chapter I.) I have come across hundreds of dilated hearts as large as a small football. There are many members of the profession who deny the possibility of these huge dilatations, one of their reasons being that the pericardium is such a very tough membrane that it would be impossible for the heart to stretch it to the extent described.

Let us glance at the facts of the case.

Professor Leonard Hill and Dr. Barnard in the Physiological Laboratory of the London Hospital, performed some experiments which were published by Mr. Barnard in the Transactions of the *Journal of Physiology* for last year. On pressing very firmly on the abdomen of a narcotised dog, they found that it was possible to completely empty the abdominal pool. They could not only void the splanchnic venous area, but they could even drive the blood through the heart and lungs into the aorta *provided that the pericardium were intact*, but if the latter were slit open, then the right heart expanded readily, retaining the whole of the blood. In this way the restraining influence of the pericardium was demonstrated.

It was also found that the heart would sustain a pressure of five atmospheres provided that the pericardium remained

intact; but when the pericardium was slit open, the heart readily dilated under a pressure of less than one atmosphere. The foregoing experiments prove beyond a doubt, that it is practically impossible for a heart to dilate provided that the pericardium be intact; but it was found that if the membrane had been recently subjected to an attack of inflammation such as in rheumatic fever all its restraining influence vanished, and it, together with the heart, was readily dilated under a pressure of less than one atmosphere, the state of inflammation having the same effect on the membrane as slitting it up.

These gentlemen regard the heart as a part of the arterial system, the endocardium corresponding to the intima, the myocardium to the muscular coat, and the restraining pericardium to the tunica adventitia.

So that it is easy to conceive the possibility of large dilatations of the heart occurring either after influenza, the zymotic diseases, or indeed any condition which might produce inflammation of the membrane. It would seem as if a sudden shock to the nerves might have the same effect as inflammation on the pericardium, as it is well known that sudden emotions of any kind may produce dilatation of the heart: *exempli gratia*, a patient whose heart I had examined half-an-hour previously and found normal, sustained a sudden mental shock. On percussing

immediately, I found the left heart dulness extended beyond the mid-axillary line.

Some years ago, in consultation with Dr. Ferrier, I saw a patient whose heart had been enormously dilated by sudden fright. The patient was suffering from recurrent syncope, which bid fair to be soon fatal. The condition, however, yielded to subcutaneous injections of strychnine and digitaline, combined with resisted exercises.

This condition seems to have been familiar to the laity at the early part of the century, as Alexandre Dumas in his work "Taking the Bastiile" says of Ange Pitou when chased by the serjeants, "His heart, *enlarged with terror*, beat three pulsations in a second."

Anyone who takes the trouble to think the matter out on these lines will not be surprised to find how very frequently the dilatations occur, and how often they lie to a great extent beneath lung tissue.

Now let us for a moment consider the physical conditions of a heart dilated to the size, say, of a small football. A heart of that size contains a considerable quantity of blood and weighs considerably more than a normal heart. The dilated heart itself does not weigh more than the normal heart, but it is the contained blood that weighs. If we consider the pericardial conditions (page 29) under which these dilatations occur, we can conceive that possibly

the pericardial ligaments which were only meant to anchor the normal heart might become stretched by the increased weight of the dilated organ, especially if, as is possible, they were somewhat affected by the inflammation which caused the dilatation.

Now dilated organs such as we have described, may flop about in the direction of gravity, and besides taking up an inordinate amount of space in the thorax and thus reducing the area of the expansion of the lungs, may exert a direct pressure on any or all of the important structures in the thorax according to the extent of the dilatation and the position of the patient at the time.

As early as 1896, March 21st, a paper of mine was published in the *Lancet* on the "Schott Treatment," in which I made the following suggestion:—

"The due consideration of the amount of space taken up in the thorax by the enlarged heart, suggests a train of thought which may perchance prove highly instructive in dealing with certain obscure symptoms, among which the following may be instanced as direct effects of intermittent pressure:—

1. Mid-sternal pain from pressure against the sternum.

2. Sense of suffocation from mechanical pressure on the lungs, vagus and diaphragm, in addition to the supervenosity of the respiratory centres.

3. Brachial pain, due to perineuritis, tingling numbness,

and coldness from pressure on the plexus and axillary artery in the space between the first rib and clavicle. This may happen, the shoulder being fixed, from the dilated heart pressing the ribs outwards and upwards. Oertel attributed the disappearance of the radial pulse during forcible expiration to the "compression of the subclavian artery by the strongly raised upper ribs." (See Von Zeimssen's *Handbook of General Therapeutics*, vol. iii., p. 468.)

4. Pain from perineuritis of the intercostals, from intermittent pressure of the dilated heart on inflamed nerve sheaths.

5. Referred pain in distant parts (see Treves' "Surgical Anatomy").

6. Pressure on vagus, causing (*a*) intermittent action of the heart by alternately stimulating and paralysing the cardiac inhibitory fibres; (*b*) gastric disorders of various kinds; and (*c*) cough, &c., from stimulation of the recurrent laryngeal nerves; in one case, severe pain in either ear supervened without any local origin, and subsided when the dilated heart was reduced, although it had previously resisted every sort of local application, suggesting interference with Arnold's nerve.

7. Interference with nutrition through pressure on the thoracic duct.

8. Obscure vascular conditions, such as angina pectoris,

possibly due to irritation of the sympathetic system exaggerating the arterial tone. I found the heart considerably dilated in a case of true angina pectoris, in which the patient complained of a severe thoracic fulness, with pain in the back and left arm before each attack.

9. Pain between the shoulders is a very common symptom in considerable dilatations; it may be shown to be partly due to backward pressure of the heart, by the pain leaving the back and establishing itself in the præcordial region on the assumption of the prone position.

10. That inframammary pain is often due to pressure of the cardiac apex against the ribs and intercostals is proved by the shifting of the pain correspondingly to the change in the position of the apex, and to its disappearance on reduction of the heart to the normal size. I have seen all these symptoms yield to the "Schott Treatment," not excepting those of true angina pectoris. In reply to those who have found the method unsuccessful and even harmful, I can only say that this has not been my experience. Either they have grasped the rationale of the treatment imperfectly, or they have been unfortunate in their selection of cases.

11. That asthma is often due to pressure of the dilated heart on the vagi, will be shown in the succeeding chapters.

It has been objected that it is impossible to have such pressure as we have described (*a*) because of the negative

pressure in the thorax, and (*b*) because such pressure cannot be exerted by a fluid tumour; but such objections are, I venture to think, sufficiently answered by reference to the well-known fact that an aortic aneurysm will eat away the sternum.

In the class of heart dilatation to which we are presently going to refer, namely, that associated with asthma, there is usually a large amount of emphysema, which implies the loss of elasticity of the lung and consequent diminution of the negative pressure. And also at the end of a deep expiration the negative pressure is minimised. (See p. 67.)

CHAPTER IV.

ETIOLOGY OF CARDIAC DILATATION.

From an examination of a hundred cases in my own practice I have compiled the following table of some of the principal causes of dilatation.

1. Irritation of the nucleus, the trunk, or of any branch of the vagus.
2. Asthma.
3. Whooping-cough.
4. Measles.
5. Zymotic diseases.
6. Emphysema.
7. Influenza.
8. Prolonged mental worry.
9. Shock. (*a*) Mental; (*b*) Physical.
10. Anæmia and Leucocythæmia.
11. Overstrain, as among cyclists, overtrained athletes, and young recruits.
12. Pyrexia.

13. Constitutional disorders.
14. Heredity.
15. Childbirth.
16. Valvular disease.
17. Old age.
18. Autotoxis. Ulceration; Nephritis.
19. Hiceough.
20. Angina Pectoris.
21. Obesity.
22. Abuse of cocain, tea, tobacco, alcohol, and opium.
23. Alterations in the blood and vaseular system.

Irritation of any part of the vagus, as in chronie dyspepsia, asthma, bronehitis, ete.

Asthma stands close to the head of the list, partly from the fact that a large proportion of the hundred eases were asthmaties, and they all had dilatation of the heart: with asthma as a cause we must inelude emphysema, as they are almost invariably concomitant.

Whooping-eough is proverbial as a cause, and the eardiac dilatations following on pyrexia and the zymotic diseases are apt to beeome chronic if the patients be allowed to get about too soon.

Influenza is a very frequent eause of eardiae dilatation, and I believe the well-known weakness after an attaek of

influenza to be partly due to this factor, probably through vagal poisoning. (S. Mackay). (See p. 99.)

Prolonged mental worry is also very prone to produce cardiac dilatation.

Mental shock as a cause is illustrated by the case of the female patient on p. 21, and by Case XIV. amongst the asthma cases in Chapter XVI. Physical shock is figured in Cases III. and IX., in the same chapter.

Anæmia and leucocythæmia are well known causes.

Overstrain is also a very frequent cause. I had an opportunity last year at Henley of examining the heart of one of the boat's crew which had just won a hardly contested race. The oarsman was in a state of extreme exhaustion and dyspnœa, so frequently seen on these occasions, and *I found the left heart dilated beyond the mid-axillary line.* I believe cardiac dilatation to be the cause of "staleness" from overtraining, as I have had opportunities of examining several of these cases and have invariably found the heart to be dilated.

Constitutional disorders such as gout, atheroma, renal disease, myxœdema, syphilis, rheumatism, we must by no means omit from our list; and as regards heredity, it is undoubtedly true that certain families are prone to acquire dilatations of the heart on the least provocation.

Severe confinements too tend to the condition under consideration, and the state of maternity is known to con-

duce to a fatty condition of the heart, which is invariably accompanied by dilatation; as are many cases of nervous disorder, *e.g.*, spastic paralysis and locomotor ataxy.

I have placed valvular disease as a cause so low down on the list partly because so few of the hundred cases referred to suffered from it, but every one of the cases that had cardiac disease had also enlargement of the heart.

Valvular Disease.—Valvular disease is, of course, a frequent cause of cardiac dilatation. Gibson says:—That in considering the morbid appearance found in cases of cardiac dilatation, it is necessary to bestow attention upon several points—the size of the heart, both in whole and in part; the weight of the organ; the thickness of its walls; and the consistence of its texture.

It is rare to find any considerable dilatation of one chamber by itself: most commonly every part of the heart is involved; but the part which is most subject to the influence at work suffers most markedly. It has been shown repeatedly that an obstruction is more likely to produce hypertrophy without dilatation, while regurgitation is more commonly followed by dilatation of the chamber into which the regurgitation takes place, attended by more or less hypertrophy.

In cases of aortic obstruction there is comparatively little tendency towards dilatation, and the chief change is more or less hypertrophy of the left ventricle, with but

little alteration in any other chamber. Aortic incompetence almost invariably produces great dilatation of the left ventricle, accompanied by a high degree of hypertrophy. In mitral obstruction there is comparatively little dilatation of the left auricle, hypertrophy in a more or less pure form being present; while in mitral incompetence there is a very high degree of dilatation of the auricle. The fact must not be overlooked that in mitral incompetence the left ventricle is very commonly—indeed, almost invariably—dilated as well as hypertrophied, the reason for which appears to be that there is in this affection such a large residuum of blood.

The right ventricle is very frequently involved as a consequence of the affections of the left side. Aortic disease does not, as a rule, produce much influence upon the right chambers, unless the left ventricle has yielded, in which case the effects are practically those of mitral disease—aortic lesions with mitral symptoms, as Broadbent terms them.

Mitral obstruction and incompetence, more particularly the latter, exercise the most powerful influence upon the right ventricle, by interfering with the onward passage of the blood in the lungs, and in these affections there is almost invariably some dilatation of the right chambers. In the lung affections, which are likely to give rise to dilatation, there may be great enlargement of the right

ventricle, usually attended by more or less hypertrophy, and it is common to find that both in affections of the left side of the heart and of the lungs, the auricle, as well as the ventricle on the right side, are dilated.

The change in size is usually accompanied by some alteration in form, but this is more especially seen when the right side of the heart is particularly affected, seeing that left-sided dilatation is commonly followed by that of the right. In aortic incompetence for example, it is a very common thing to see hearts enormously enlarged from dilatation of every chamber, while the relative proportion of each part of the heart is but little modified. In mitral incompetence more particularly, the heart does undergo some alteration in form, since the left auricle and the entire right side of the heart are more likely to be modified than the left ventricle.

When dilatation results from any pulmonary condition, the right side of the heart may be almost solely affected, and it is sometimes so extremely large as to entirely overshadow the left. In such a case the right ventricle often forms the apex of the heart, and constitutes the vast mass of the organ. The size of the chambers has already been given in the anatomical section, but it may be stated here that the average measurements of the ventricular cavities, taken from the apex of the cavity up to the base of the nearest sigmoid cusp is, according to Hamilton, on an average: for

the left ventricle, $3\frac{1}{4}$ inches in man, and 3 inches in women; while those of the right ventricle average $3\frac{3}{8}$ inches in men, and $3\frac{1}{16}$ inches in women. These measurements are often greatly exceeded in dilatation.

The weight of the heart in simple dilatation is not increased. This, however, is to be regarded as a theoretical speculation more than a scientific fact, since some hypertrophy is almost invariably an accompaniment of dilatation.

The auriculo-ventricular orifices undergo changes in dilatation of the cavities, and their dimensions are considerably increased. As a consequence the cusps are rendered incompetent. This is not only the result of an enlargement of the orifices, but it occurs in consequence of dilatation of the cavities, by means of which the attachment of the papillary muscles to the walls is removed to a greater distance than in health. Sometimes this is to some extent obviated by degenerative processes in the papillary muscles whereby they do not contract, and, therefore, allow greater possibility of the cusps meeting. Both fatty and fibrous changes in the papillary muscles may effect this change. If there be much dilatation of the auricles, there is usually considerable distension and enlargement, both of the auricular appendices and of the great venous channels.

In simple dilatation the walls ought necessarily to be somewhat thinner than in health. This, however, may be very safely stated never to occur. As has already been

shown, the thickness of the left ventricle is, according to Hamilton, on an average about a quarter of an inch at the apex and half an inch at its thickest part near the base, both in men and women; while the right ventricle is on an average one-eighth of an inch thick in both sexes; but parts of the right ventricle were found by this observer to cover sometimes a quarter of an inch, or as little as one-sixteenth.

The conditions commonly associated with dilatation require but little comment. Many of them are etiologically linked with the dilatation, others however are its results; but as these latter are essentially produced by venous stasis, there is no necessity for following them out in this place.

Age is also a factor which must be considered. Gibson says, dilatation is more common at two epochs of life than during the remainder. During the period of adolescence, when all the different functions are undergoing adaptation, dilatation is more easily induced than during the prime of life. Again, in elderly people, in consequence of the incidence of atrophic and degenerative processes dilatation again becomes more common. Occupation plays also an important part: those who are engaged in work which entails considerable physical stress are much more liable to cardiac dilatation than those whose occupation does not expose them in this way.

Autotoxis is a frequent cause of dilatation. The seat of self-poisoning is often to be found in connection with decayed teeth, erosion of the cervix uteri—a very frequent cause in hysterical cases—and in nephritis, from retained excreta.

Hiccough.—I was called to the West of England some years ago to a patient who had been suffering for nine months from hiccough which had apparently become a habit as in cases of trade-spasm. I found the heart considerably dilated and could trace no other cause for the dilatation except the hiccough. I employed a simple means of arresting the spasm; it was imparted to me by a former patient, and I am bound to say that I have never known it to fail, and if the theory that hiccough is due to spasm of the diaphragm be a correct one the *modus operandi* of the method is obvious. It is as follows:—Direct the patient to close the anterior nares with the third fingers and to occlude either external auditory meatus with a thumb. Now direct an attendant to hold a glass of water to the patient's lips and let him slowly sip it down. The case referred to was simply dying from exhaustion, and every possible means, including chloroform, had been tried to relieve the condition. The water plan, however, was efficacious; the spasm was interrupted and *did not recur*.

Obesity is an obvious cause of dilatation, from the fact

that it entails so much extra work on the heart to supply the large amount of fat distributed throughout the body with blood, and that the organ itself is hampered by accumulations of adipose tissue which naturally tend to throw it out of gear.

Any state which tends to alter the conditions of the vascular system, such as gout, syphilis, angina pectoris, and arteritis obliterans. I saw an example of the last in consultation with the late Professor Charcot, where there was considerable dilatation of the heart, but whether the narrowing of the lumen of the vessels was cause or effect is open to conjecture. (See chapter on Angina Pectoris.)

CHAPTER V.

SOME SIGNS OF CARDIAC DILATATION.

1. Inereased area of pereussion.
2. Apex beat defleeted outwards.
3. Feeble first sound.
4. Inereased seeond sound.
5. Albuminuria assoeiated with enlargement of the kidneys.
6. Enlarged spleen.
7. Enlarged liver.
8. Anæmia of the various organs—characterised espeeially by loss of memory, amblyopia, and baldness; dislike to exertion; numbness of one or both arms.
9. Tendency to fatty degeneration and early areus.
10. Pulse feeble and intermittent, eompressible, usually frequent, but in cases of large dilatation often slow.
11. Œdema, especially of the lower lids and ankles,

also general anasarca, sometimes well marked on the chest.

12. Baldness.

13. Thyroid enlargement.

Increased area of percussion.—It is extraordinary to what dimensions the dilated heart may attain. It is not at all uncommon in a fat patient to find the left heart margin well beyond the mid-axillary line, but dilatations of the right side of the heart are far less common. In these cases of extreme left heart dilatation the apex beat may be well made out by directing the patient to lean forwards, and feeling for it with the point of the index finger, especially during expiration.

The apex beat may be deflected outwards and upwards, and very frequently outwards and downwards, pushing down the diaphragm. I have frequently been able to feel it in the epigastrium.

Feeble first sound.—The first sound is liable to be very feeble in large dilatations, on account of the decreased force of the muscular contractions of the cardiac wall; indeed, in the case of one of my patients who had a very large fatty heart it was hardly to be made out at all; whereas the second sound is apt to be emphasized.

This is probably due to increase of resonance, owing to the enlargement of the cardial cavity on closure of the aortic valves.

Albuminuria, too, is a very frequent occurrence; it rapidly disappears on reduction of the cardiac diameters. In these cases the kidneys may be readily percussed out from the back with my plexímeter. An increase in the size of these organs is invariably present; this diminishes on the disappearance of the albuminuria.

Enlargement of the spleen is also a very common sign of cardiac dilatation; people living in malarious districts, are prone to the enlargement of both these organs.

The liver almost invariably suffers, becoming congested and enlarged from lack of vis a tergo.

General anæmia is also very apt to occur, including anæmia of the special organs and viscera. The brain being, so to speak, on the top floor, is very apt to suffer from anæmia, and the anæmia is characterised by loss of memory, loss of concentrative power, amblyopia, occipital headache, feeling as though the top of the head would come off, and sometimes slight paralysis, first of the right arm, then of the left, as in cerebral anæmia. The left side of the brain is usually affected first. This must not be confounded with slight paralysis of and pain down the left arm. Much earlier symptoms of cardiac dilatation are due to perineuritis from direct pressure of the dilated heart on the brachial plexus. (See p. 24.)

Tendency to fatty degenerations.—Fatty degeneration is

due to a local cutting-off of the blood supply. Now the enfeebled heart does not drive sufficient blood anywhere, therefore cardiac dilatation tends to special local fatty degenerations especially of the heart itself, whose walls receive an insufficient supply of pabulum through the coronary arteries. In advanced cases of fatty infiltration the cardiac margins are very frequently wavy.

Pulse.—That the pulse is feeble is of course obvious; it is also apt to intermit, and in some cases become very frequent. I have often observed a pulse of 140, and on several occasions I have been unable to count it, without any pyrexia being present; but I have observed, on the other hand, that in very large dilatations, the pulse sometimes sinks to 30 and even lower.

Œdema is an almost invariable sign in the larger dilatations of the heart; it is of course due to want of cardiac energy. It is very apt to show itself in the lower lids and ankles, but it is sometimes general, and I have frequently observed œdema of the chest characterised by pitting on pressure, especially in fat patients whose hearts were greatly dilated. The patients often present a lack of expression suggestive of myxœdema. Œdema of the breast in women is a specially characteristic feature, and on its disappearance the nipple, which had been supported by the œdematous condition, deflects outwards and downwards

only to move upwards and inwards when the gland resumes its normal texture and health.

Billings says, "that an œdema of the angio-neurotic type has been described in the hands and arms in asthma, and I have frequently observed this phenomenon in that class of cardiac case, which is associated with dilatation."

Baldness is also sometimes concurrent with cardiac dilatation. I have frequently noticed a growth of hair following on the reduction of the cardiac dilatation, and consequent increase of blood-supply to the hair-follicles. (See Case XXXIV.)

The diaphragm may be so much depressed by the dilated heart, that the apex beat may be felt even in the epigastrium.

Enlargement of the Thyroid gland.—I have known a case where the gland resumed its normal size simply on the reduction of the cardiac dilatation.

Speaking generally a dilated heart does not half do its work; thus you may have at once anæmia and congestion of any part of the body; anæmia from insufficient blood-supply, and from diminution in the number of the red blood corpuscles due to suboxidation, itself owing origin to the feeble flow through the capillaries of the lungs, as well as to congestion due to stasis of what blood there is, owing to lack of vis a tergo. These patients, therefore,

often have a capillary congestion and high colour over the malar regions, giving them an air of robustness, and further, the general œdema enhances the delusion. As their friends are apt to say: "Their looks don't pity them," and they frequently get shelved as hysterical, whereas in reality they are in a pitiable plight. Many such cases have been brought to me as cases of hysteria, but on reduction of the cardiac dilatation they have become again as their friends would have them, and we have heard no more of their hysteria.

CHAPTER VI.

SOME SYMPTOMS OF CARDIAC DILATATION.

1. Lassitude.
2. Want of stamina.
3. Tachycardia.
4. Præcordial pain.
5. Pain, numbness or tingling in one or both arms.
6. Cold extremities.
7. Dyspnœa, especially on exertion.
8. Headache.
9. Loss of memory and inability to concentrate thoughts.
10. Amblyopia.
11. Indigestion.
12. Constipation.
13. Asthma.

14. Biliousness.
15. Tendency to faintness.
16. Depression.
17. Irascibility.
18. Angina pectoris.

Speaking generally, we may have the symptoms due to disordered functions of any organ of the body owing to want of sufficient blood-supply to that organ, in addition to those produced by direct pressure of the dilated organ. (See effects of the pressure of the dilated heart, Chapter III.) Lassitude and want of stamina are very frequent symptoms, whilst tachycardia is more frequent in comparatively small dilatations than in greater ones. Præcordial pain is fairly constant, whilst pain down the arms, numbness, and tingling in the fingers are often felt. (See Chapter V.—Anæmia.)

Coldness of the extremities is a very common symptom, whilst dyspnœa on exertion is nearly invariable.

Headache, especially of the anæmic type, is usual, whilst loss of memory is fairly common, and I have seen several cases of amblyopia due to brain-anæmia recover their power of vision when the heart became normal. These cases had usually consulted many eye-specialists in vain.

On indigestion and constipation it is needless to com-

ment, while asthma will be fully considered in the chapters devoted to that subject.

Biliousness, depression, and irascibility follow on the congested liver usually present, whilst tendency to faintness and even syncope are obvious symptoms.

Angina pectoris will be discussed in a subsequent chapter.

We constantly see recorded in the papers sudden deaths from shock, either mental or physical, in which the doctor has said "there was nothing wrong with the heart." Such a case occurred last week in the case of a man who was acting as second to a prize-fighter. Now cardiac dilatation tends to the production of emphysema, which is due to the dyspnœa which accompanies heart-dilatation on exertion. In many cases, the enlarged heart lies behind the lung and so escapes notice; thus the patient, although walking about, apparently with a normal heart and in good health, is in imminent danger of death, owing to the fact that the dilated heart is unable to respond to any sudden call upon it, as in mental and physical shock. The prize-fighter's second above alluded to, was intensely interested in the contest, for he had put more money than he could afford to lose, on the loser. The emotion thereby engendered was too much for his heart, which consequently stopped beating. *Now this individual would undoubtedly have been passed as a first-class life at an insurance office,*

as the dilatation could not have been made out by the ordinary methods of percussion. Such cases abound and have not necessarily any cardiac symptoms.

This man was supposed to have died from failure of the heart.

CHAPTER VII.

TREATMENT OF CARDIAC DILATATION.

Let us consider the treatment of cardiac dilatation under the following heads:—

1. Removal of the cause.
2. By rest.
3. By drugs.
4. By inhalation of oxygen gas.
5. By exercise—(*a*) hill-climbing, (*b*) cycling.
6. By breathing exercises.
7. By special treatment.

The removal of the cause of the disease is an obvious factor in any line of treatment, and needs no comment. Rest is most valuable in the treatment of this disorder. The term rest should include both mental and physical rest: the mere fact of resting often *ipso facto* removes the cause of cardiac dilatation—as, for instance, when that dilatation has been brought about by mental or physical strain. Rest, too, in the horizontal position, relieves the

heart of much of its work, and, up to a certain point, allows considerable scope for the exercise of *vis medicatrix naturæ*, and there is no doubt that in many instances the patient's condition will be greatly ameliorated by being kept in bed for a lengthened period, and by being protected from mental worry. But it is precisely this kind of rest that is so difficult of achievement, and further, in the majority of cases, it is not far-reaching enough. The heart is an organ which is essentially made for work, and while rest will frequently remove many of the symptoms of cardiac dilatation, it usually fails to reduce that dilatation, and on the resumption of ordinary occupations the dilatation is very apt to recur. Very little is known concerning the branched fibres of the myocardium, but that they are made for work is obvious, that from the moment our hearts begin to beat till the day of our death, they never cease to exercise their functions. Having these facts in view, it was Stokes, of Dublin, who first suggested exercise as a treatment in heart lesions.

As regards treatment by drugs, we have several very valuable remedies to hand, and at the head of these we must undoubtedly place digitalis, which so often tends to contract the heart and steady its action. Its effect on tachycardia, especially in doses sometimes as small as one minim, is notorious. Strophanthus, too, forms a valuable ally, and is often tolerated when digitalis is not. Then

stimulants are often of use, such as ether, brandy, with hot applications to the præcordium. But perhaps the most potent cardiac tonic is strychnine; it may be given by the mouth, though it acts better when injected hypodermically. Vaso-dilators too, to wit, nitrite of amyl and nitro-glycerine, give temporary relief to the heart by dilating the blood-vessels, whilst iron and arsenic give tone to the heart by relieving the anæmia, which almost invariably accompanies cardiac dilatation.

Thyroid extract is sometimes of great value; but it should be given very guardedly, and never to young, thin, neurotic patients, as it is apt to produce untoward symptoms such as tachycardia, and if pushed, even Graves' disease. The class of case in which it is useful is mature age, with an abnormally slow pulse and general œdema, somewhat simulating the myxœdematous condition. (See Chapter V., page 41.) I also find the Vibrator of Liedbeck (Stockholm) of great service in the reduction of congestions of the liver and other organs. The instrument may be obtained from Mr. Geere Howard, Electrician, Berners Street, W. Its utility has been greatly enhanced by Dr. Herschell, who has devised a method of connecting it with the main electric supply, by means of a wall-plug.

The only drug I give as routine practice is a mixture containing peptonate of iron and manganese made up in old port. It is to be obtained at Orchard's, Chemist,

Salisbury; he made it up years ago to my prescription. I have never known it to disagree with a patient who requires iron, and it has the following advantages:—

1. It does not upset the digestive apparatus or cause constipation.
2. It does not discolour the teeth.
3. It is pleasant to the taste, and is taken as a liqueur three times a day after meals.

I wish merely to touch on drugs, for details my readers are referred to Standard works on therapeutics.

But there is one factor which is very potent indeed in the class of case under consideration, and that is the frequent inhalation of pure oxygen gas. Patients with dilated hearts all suffer from sub-oxidation of the blood, and the inhalations of oxygen gas not only act as a general stimulant, but allow of an increased amount of oxygen being absorbed by the red blood corpuscles during their passage through the capillaries of the lung. This is shown by the fact that the exhibition of oxygen gas relieves the following symptoms. The patient sits up in bed, gasping for breath with cardiac dyspnœa, the pulse feeble, the skin cold and clammy, and the lips cyanotic. After a few whiffs of the gas, the pulse will improve, the dyspnœa will be very much diminished, and the lips resume a normal hue. Having regard to the value of the gas inhalations in cases of stress, it is my practice to give it two or three times a

day in cases of chronic heart-disease, with obvious benefit to the patients. But we must just sound one note of warning. Human nature has a lamentable tendency to abuse everything, and I am told that there is already such a thing known as the "oxygen habit."

Certain breathing exercises are specially useful for fully expanding the lungs, after they have been hampered in their action by the dilated heart. These are fully described in Campbell's work on *Respiratory Exercises in the Treatment of Disease;* they will be further alluded to in the chapter on "The Treatment of Asthma."

We have already alluded to the treatment of heart complaints with exercise, first suggested by Stokes, of Dublin; but Stokes did not live to completely formulate his plan of campaign, it has been left to others to carry out his original idea.

Stokes was followed by Oertel, who advocated graduated hill-climbing; and by Beneke, who found that the baths at Nauheim reduced the cardiac dimensions, and he even went so far as to claim that the baths promoted the absorption of cardiac vegetations. But it is to the Brothers Schott, of Nauheim, that we owe the genesis of the treatment now known as the "Schott Treatment" or "Nauheim Treatment." It consists in a combination of the Nauheim baths and certain "exercises with resistance" (*Wiederstands Gymnastik*) devised by the Brothers Schott. It

has been said that these are merely a copy of the Swedish exercises of Ling. But this is not the case. Dr. Schott tells me that he and his brother were not acquainted with the Swedish methods, when they elaborated their Wiederstands Gymnastik.

This treatment should then obviously be known as the Schott treatment, and not the Nauheim treatment. For a full description of these methods I refer the reader to a paper read by Dr. Hyde on the Nauheim Treatment, before the British Balneological and Climatological Society, and published in the Society's Journal for October, 1898.

I will not attempt to explain the intricate nervous phenomena by which these methods attain their end (See Harveian oration on Circulation, by Lauder Brunton); but I believe that the stimulation of the skin by the billions of gas bubbles contained in the carbonated baths, has, at all events, something to do with the matter. In 1889, when I was at Bignasco in the Canton Ticino, recovering from a bad hunting accident, I had a slight dilatation of the heart. The Falls at Bignasco come over a rock cliff nearly 300 feet high, and the basin, which has a rough rock bottom, reminds one of a gigantic bath of soda water. I used to anchor myself twice daily in this basin, with the result that the cardiac dilatation disappeared, and I again became well and strong after using the bath for a week. Previously to this I had been for three months

in a state of extreme debility. My recovery, of course, may have been due to natural causes, but I am not inclined to think so. I had no bias towards this idea, as I was then unacquainted with the Schott methods. With regard to the exercises, I believe that one factor at least in the reduction of cardiac dilatation is a very simple one, namely, the free flow of blood in the veins, from the fact that the muscles which come into play during the resisted exercises are firm and free from irregular contractions, so that possibly they may act as supports for the vessels, in the same way as a cement bed does for a drainage pipe under ground. Neither the baths nor the exercises are supposed to act directly on the heart, but they relieve the over-distended organ by dilating the blood-vessels. I have no doubt in my own mind that these methods should stand at the head of the list of methods of treatment for cardiac dilatation; but no wise man will confine himself to any one line of treatment. The best results will be obtained by a judicious combination of rest, drugs, and exercise, with the Schott methods, according to the exigencies of each case. I have not referred to hill-climbing and cycling as they should obviously be practised as a *nach Kur* (or after-cure), when the heart has resumed its normal dimensions. They will be considered later on.

I will now proceed to describe briefly my own method of conducting the treatment.

I have found from experience that if you wish to obtain good results, it is necessary to pay the greatest attention to details, and that there is no symptom which may supervene, however apparently insignificant, which may not be of vital importance. Keeping in mind the radical changes which you propose to make (the heart may be reduced as much as one inch in half-an-hour by exercises alone) it is advisable to treat each case as though it were a case of severe illness. It is, therefore, my practice to visit the patient every day for the first week or ten days, then every other day, and finally twice a week during the month or six weeks which is the average duration of the course. Since I have taken to this line of practice, I have obtained much better results. Although undergoing special treatment, the patient requires ordinary medical supervision; and such symptoms as headache, indigestion, constipation, etc., should, of course, be promptly attended to. To relieve the chronic congestion of the liver, which is usually present, I am in the habit of prescribing euonymine and iridin, of each a grain, made up with extract of lettuce, and adding a quarter of a grain of podophyllin should the bowels be confined, in addition to ordering the patient to eat a raw apple at bed-time, at the same time attending to the diet generally.

For the baths I import the mother salt from Nauheim, and find Sandow's tablets, which are sent out in boxes to-

gether with bags of bicarbonate of soda, very convenient for forming the carbonic acid gas. Each box contains eight tablets of hydrochloric acid and four bags of bicarbonate of soda, so that it is easy to make a subdivision for the varying strengths of the baths. Three preliminary baths are given, consisting of ten pounds of the Nauheim salt to fifty gallons of water. It is inadvisable to commence with the carbonated baths, as they are sometimes apt to induce severe muscular tremors which may be mistaken for convulsions by the patient and friends. The first carbonated bath should consist of the plain salt bath as described *plus* two hydrochloric acid cakes and one bag of bicarbonate of soda. A bath sheet is placed on the surface of the water, completely covering the patient with the exception of the head and the neck. This sheet serves three ends: (1) it maintains the *equus animus* of the patient; (2) once it is wet, it imprisons the carbonic acid gas, which would otherwise escape; consequently it increases the gaseous pressure and therefore the efficacy of the bath; (3) it prevents the patient from inhaling the carbon dioxide, which otherwise is possible, for although the specific gravity is greater than that of air, the patient's mouth is almost on a level with the surface of the water. The inhalation of CO_2 is apt to produce headache, and the nurses are usually instructed to fan the patient vigorously to obviate this. But the sheet method

is simpler and does not alarm the patient. I now seat myself at the bathside, and keep two fingers on the radial pulse until it is time for the patient to emerge. The pulse having been taken in a recumbent position before the bath, we find it usually quickens a few beats from the effort of getting into the bath. In a few minutes it begins to slow down, becoming fuller and softer; when it begins to rise again the patient has had enough.

Now there is just one moment, and one only, when the patient has derived the full benefit from the bath, and that is when the pulse is on the turn from its lowest rate to a quicker one. In the event, however, of the pulse being abnormally slow, it is well to reverse this order. It would seem as though the bath would tend to bring the pulse to the normal in any case, namely, if it be too frequent it tends to reduce the number of beats, whereas, if it be too slow, to bring it up to the normal; so that in the latter case you take the patient out of the bath when it begins to slow. Now, unless you observe the pulse all the time, you are very apt to miss the happy moment, and I can assure you that if you do so it will make a vast difference in your results. Further, you are present in case the patient has any misgivings, which is frequently the case at the commencement of the treatment. After the patient has been in the bath for two or three minutes, I instruct the nurse to administer one or two bags full

of oxygen, the inhalation of which, has the three-fold object: (1) of stimulating the patient and obviating the occasionally depressing effect of the bath; (2) of hyper-oxygenating the blood; and (3) of counteracting the effects of possible small inhalations of carbonic acid gas.

We wait until the patient has been a short time in the bath before giving the oxygen, as then the respirations become deeper and the pulse becomes fuller and stronger, so that an increased amount of blood is passing through the lungs, whereby the oxygen is more readily taken up by the red blood corpuscles. After the bath the pulse should become full, slow and soft to the touch, a condition which I have called "velvety." I can only say that I have obtained better results since the exhibition of the oxygen. The baths are increased at the following rates, namely, a quarter, a half, three-quarters, and full at intervals of every sixth bath, and the full bath continued to the end of the course. Only three baths are given running, every fourth day there is no bath, but two sets of exercises. After the bath, from which the patient should never be allowed to get out unaided (as any exertion immediately after, either bath or exercises, diminishes the good effect), the patient should be enveloped in a well warmed bath towel and briskly rubbed with a light hand, but on no account allowed to dry him or herself, and should then be immediately put to bed, between blankets, and if feeling

cold should have a hot bottle applied to the feet, and should remain in bed for an hour. It is not well for patients with anæmia of the brain to read during this hour; they should be encouraged to sleep. The plain salt bath should be given at a temperature of 95° Fahrenheit, and gradually decreased as the strength of carbonic acid is increased to 85° or even 80° in the summer; but there should be no absolute law about this, the circumstances should be varied to suit each case. As a rule the colder the bath, the more bracing the effect. Patients will often complain of the coldness of the stronger baths, but this passes off after the gas bubbles have been acting on the skin for a few moments. On no account should the bath be given within an hour of a meal, and I generally prefer that the bath be given in the morning and the exercises in the evening; but many patients prefer the bath in the evening in order to make it fit in with dressing for dinner. The bath should be omitted on every fourth day, otherwise the patient is apt to become "over-bathed," that is to say, the bath gives rise to great discomfort, and the pulse, instead of getting slower, tends to become more frequent.

The exercises are given once each day and twice on non-bathing days, the patient resting an hour after each set under the same conditions as after the bath, with the exception that, as it is not necessary to undress, they need not go to bed.

The diagnosis should be very carefully made, and a chart taken of the exact size of the heart for future reference. It is my custom to take the chart once a week, thus I can see how the case is progressing. This chart is only for the benefit of the medical attendant, and it should not be allowed to pass into the possession of the patient at the end of the treatment, as it may be flaunted about amongst friends and so bring the methods into disrepute. Sir Richard Douglas Powell told me the other day of a case he had heard of, where the chart was handed round as an entertainment at a tea-party.

Speaking broadly, the class of case which is likely to benefit from the treatment is that which consists of, or is dependent on, derangements of the circulatory system. It is well to bear this fact in mind, because considerable discredit has been thrown on the Schott treatment, it being said that its votaries claim too much for it. What it does, in a word, is to dilate the blood-vessels, and so reduce the enlarged heart.

Although it is exceedingly inadvisable to rashly prescribe a new treatment with which one is unfamiliar, yet it is one's obvious duty to one's patient to put oneself to the pains of at least thoroughly investigating that treatment; and it is also possible to err on the side of extreme caution. This is well exemplified by the following quotation from *The Action of Medicines*, by Lauder Brunton.

"Under the influence of the baths and of the movements, you get extraordinary results. I was a good deal astonished a little while ago, when I recommended a patient to go to Nauheim, to receive a letter from the general practitioner who had been in attendance upon him, saying that the Nauheim treatment was too new to enable him to form any opinion about it: that he did not think it was any good and, therefore, he advised the patient not to go. The man in question simply knew nothing about it: he had not taken the trouble to enquire, and so he decided upon a basis of pure ignorance."

We now come to the after-treatment, which is of the greatest possible importance to prevent the heart from re-dilating, for although that organ be reduced to its normal size it is not, so to speak, "in training," and its strength has gradually to be built up. The real cure of the patient begins after the special treatment is over—after the cause, that is the cardiac disorder, has been removed. Any sudden call on the heart might re-dilate it at this stage; and it is most important to give the patients strict injunctions as to the manner of life they should lead, for at least three weeks afterwards. It is inadvisable for a patient to go straight back to business or to worry of any kind; I find that patients do better at the seaside after being treated in London.

The patient should be instructed to practise self-resisted

exercises, by which is meant exercises in imitation of those given by the nurse; that is, by setting different sets of muscles to work in opposition to each other. This result is brought about by holding the limbs as rigid as possible, or the patient may use the Largiader's Exerciser, which gives resistance without jerk (for illustration see end of the book). Schott says that it is not advisable to employ any mechanical appliance during the actual treatment, as it is essential to have a trained and intelligent exerciser in order to know when to desist. There are instances of patients having done harm to themselves, by practising these exercises with mechanical appliances unattended by an expert. I find three or four exercises, followed by the recumbent position, for a quarter of an hour daily, are usually sufficient; they should be kept up for two or three months after the treatment proper is over. The resisted exercises should have the refreshing effect of "a good stretch," which they much resemble.

Very slight dilatations of the heart may produce considerable constitutional disturbance: these often occur in strong, active men who lead a busy life, and would be unable to afford the time to undergo a regular course of treatment, even were it necessary. This condition is characterised by a lack of energy rather than by any definite symptoms. The patient will say "I know there is nothing the matter with me, doctor, but I wish you

would just overhaul me, as I don't feel quite up to the mark." If on making a physical examination I find there is a slight dilatation of the heart, say of from half an inch to one inch, unaccompanied by any complications and the patient be otherwise strong and well, I consider it safe to advise him to purchase a Largiader Exerciser, and carefully regulating the weights, to instruct him to perform two sets of exercises daily. I was consulted by such a patient two months ago, whose left heart was dilated a little more than an inch, and who succeeded in reducing the dilatation in a fortnight by this means.

The patient, during the after treatment, should be directed to avoid the cause which originally produced his heart-trouble, he should take exercise twice a day, at first on the flat, then on gentle slopes, and lastly up steeper ascents, to give tone to the myocardium. I also recommend cycling on the flat at first, and find that my patients improve rapidly on it. My reasons for prescribing it are that the patient obtains (1) Exercise without having to sustain the body-weight; (2) Exhilaration from moving quickly through the air; and (3) as the feet are continually pushing the pedals, the movement somewhat resembles those of the resisted exercises. I find that the patients do very well on it, even when slight valvular disease is present. Dr. Schott argues that cycling is bad, being prone to produce dilatation; I do not think his

premises are sufficient, as he based his judgment on the examination of one case. He sent me a pamphlet published in 1897, which contained a skiagraph which showed a cardiac dilatation which had been produced by a long ride over a stony road. I do not think that that is a sufficient reason to condemn cycling, as it seems to have been a case of over-cycling, which is a very different matter. The result of *that* we are quite familiar with, under the name of "bicycle heart."

After trying a large number of watering-places on the south and south-east coast, I have selected Eastbourne, as a suitable place for the after-cure. I cannot always get my patients to go there, but for some reason or other, those who do, seem to be greater successes than those who go anywhere at haphazard.

The apparent advantages of Eastbourne are: (1) That it is near London; (2) that there is plenty of accommodation; (3) that the air, though bracing, is light, resembling somewhat the air on the North coast of France; and (4) that in Beachy Head is afforded an opportunity of graduated hill-climbing, *terrain kur*, which is unsurpassed. It has the additional advantage of there being a hotel at the top, so that the patient can, if he wish it, go up in the morning, have his luncheon comfortably, whilst breathing the fine mixture of down-and-sea-air, which the situation affords: further, if fatigued,

he can telephone for a carriage to take him home. I generally pass my patients as sound, when they are able to walk to the top of Beachy Head and back without fatigue or dyspnœa.

Contra-indications for the Treatment.

The Schott Treatment is contra-indicated in:—

1. Acute diseases.
2. States of pyrexia.
3. Sclerosis of the vessels.
4. Some cases of aneurysm.
5. Advanced Atheroma, especially if associated with cardiac hypertrophy.

If the treatment be applied to cases of thoracic aneurysm, it should at least be conducted with extreme care, Dr. Schott tells me that he has had some good results; but I am unable to speak from experience, as I never had such a case under my care; but as the effect of the treatment is somewhat that of the iodides on the vessels, it is possible that it might be of use in aneurysm.

Cases of dilatation, with pericardiac adhesions, also require care, as exemplified by Case IV.

CHAPTER VIII.

SOME PHYSIOLOGICAL PRINCIPLES CONNECTED WITH THE ELASTICITY OF THE LUNGS AND PULMONARY SUCTION.

Before proceeding to the consideration of emphysema, which will be the subject of the next chapter, it will be well to define some physiological principles connected with the elasticity of the lungs and pulmonary suction. This is the more necessary for the proper appreciation of the theories propounded in this work, inasmuch as it has been objected that it is impossible for the dilated heart to exercise pressure on the vagi on account of the negative pressure in the chest. As will be gleaned from the following pages, this pressure is very slight at the end of each expiration; besides, in any case, it is a question of degree. It is possible to have a force which is greater than elasticity of the lungs, and can therefore overcome the negative pressure, as exemplified by aneurysm of the aorta eating away the sternum. Harry Campbell says on this score: that the lungs occupy that part of the thorax not occupied by the mediastina, but they are not large enough to fill this space without a considerable stretching of the elastic fibres with which they are richly furnished, and hence they are ever tending to contract, this tendency increasing during inspiration and during contrac-

tion of the bronchial muscles. The pressure of the atmosphere within them, however, counteracts the tendency and keeps them closely applied to the chest-walls, heart, aorta and other contiguous structures. When the chest is opened after death, and the atmosphere allowed to press upon the outside of the lungs as well as from within, they necessarily undergo considerable contraction. Hence, we must think of these organs as ever striving to break away from their surroundings, and as thus exercising a negative pressure or suction upon them. We may speak of this as *pulmonary suction*, and it is owing to it that the pressure on the pleuræ and pericardium is negative.

From the foregoing remarks it will be clear that the lungs in no sense *support* the chest-walls, but, on the contrary, tend to suck them in. It is only towards the end of a deep expiration, or when an expiratory effort is made with closed glottis, that they exert anywhere a positive pressure. In the latter case they may be made to compress the heart and great blood-vessels with such force as actually to stop the circulation.

The diaphragm, as the most yielding portion of the chest-wall, is the part most influenced by pulmonary suction. The pressure on its abdominal surface is almost always positive. Hence, in its passive state, the diaphragm is sucked upwards into the dome-shaped form, and every contraction and consequent flattening of it has to overcome pulmonary elasticity.

After the deepest possible inspiration has been taken, the diaphragm tends to be more than usually elevated owing to the increase in pulmonary suction.

The elastic force exerted by the lungs at the end of an ordinary expiration is equal to a column of from 5 to 7 millimeters of mercury, for if after death, when the chest is in a position of ordinary expiration, a manometer be fixed in the trachea, the mercury rises to about this height on the chest-walls being punctured and the lungs thus permitted to collapse.

When the chest is artificially inflated to the position of ordinary inspiration, the mercury rises to about 10 millimeters, and the rise increases to 30 millimeters if the chest be distended to the position of extraordinary inspiration. At the end of an extraordinary expiration elastic force is absent, the lungs then being actually smaller than after removal from the body.

Hence, the elastic force or suction exerted by the lungs under varying degrees of expansion is as follows:—

At the end of an extraordinary expiration, 0

„ „ ordinary „ 5 mm. Hg.

„ „ „ inspiration, 10 „

„ „ extraordinary „ 30 „

The elastic force exerted by the bronchial muscles, when contracted, is estimated at from 1 to 2 mm. Hg.

Other aspects of pulmonary suction have now to be considered.

All the tissues of the body tend to become less elastic with advancing years. It is for instance, owing to loss of elasticity, that the skin becomes wrinkled with age; it gets permanently stretched, and, no longer tightly adapting itself to the underlying structure, is thrown into folds. As with the skin, so also with the lungs. Their elasticity, and consequently the suction they exert, diminishes much more rapidly in some cases than in others, so that while in one way we may find prematurely senile lungs at fifty, in another elasticity may be well preserved at seventy. It may also be diminished by disease. All organic diseases of the lungs—pneumonia, bronchitis, phthisis—diminish it. In emphysema, the diminution is a characteristic feature, it plays a prominent part in its pathology. Elasticity is also probably always diminished in protracted fever; Cohnheim finds it absent in typhoid; Perls has found it diminished in typhoid, typhus, and diphtheria; and both have noticed its loss in phosphorus-poisoning.

Undue stretching of the pulmonary alveoli is another very potent cause of diminution or entire loss of normal resiliency. It is in this way that effort, such as coughing and lifting weights, diminishes elasticity. Chronic dyspnœa diminishes it in the same way—namely, by causing over-expansion of the lungs—hence also the injurious tendency of all exercises causing breathlessness, such as running rapidly upstairs, swimming for a long time under water, &c.

It is seldom, however, that pulmonary elasticity is so completely lost as to do away with all suction. It is probable that this occurs only in the most advanced cases of emphysema. When a portion of the lung is solidified, as in pneumonia, it necessarily loses all elasticity, and therefore all suction. Post-mortem examination gives no evidence, however, that the parts thus solidified press upon the chest-walls, as from their distension we might perhaps expect them to do. This is because the thorax over the affected region expands, and I have no doubt that Sir Richard Douglas Powell is right in attributing this expansion to loss in pulmonary elasticity. He holds that "as the inflamed lung increases in bulk, the thoracic wall retreats," owing to the removal of pulmonary elasticity, which normally causes the ribs to be sucked in beyond the neutral point. I would suggest that a more important factor even than this is the overaction of the inspiratory muscles. This overaction necessarily results from the diminution of suction, and is, moreover, predisposed to by dyspnœa.

Effusion of gas or liquid into the pleura, diminishes pulmonary suction; the stretched elastic fibres of the lung becoming more and more lax as the effusion increases and the lung shrinks, it continues to diminish until the shrinkage of the lung has proceeded to the point at which the pulmonary fibres are no longer stretched, when it completely disappears. The continuance of effusion beyond this point causes the pressure in the pleura to become

inereasingly positive. In one ease of hydrothorax, Leyden found the pressure of the pleural fluid to be 28 millimeters Hg., and in a ease of pneumothorax, he found the pressure of air in the pleura to vary between 5 and 10 millimeters Hg. In pleuritic effusion, the heart is displaeed to the opposite side before the diaphragm is thrust downwards; for direetly the pulmonary suetion of the affeeted side diminishes—and this oecurs at the very beginning of the effusion—the heart will be drawn over by the greater suetion of the opposite side, but the pressure on the abdominal aspeet of the diaphragm being positive, it will not be until the pleural pressure beeomes still more positive that the diaphragm will be thrust downwards. Henee, as Douglas-Powell observes, "displaeement downwards of the abdominal viseera is a late phenomenon in pleuritic effusion," and "the stomach note may be obtained at the sixth rib in the nipple-line in the presence of a large effusion on that side."

In gaseous effusion into the pleura the heart is immediately sueked over to the unaffeeted side, but the pressure cannot be sufficiently positive to displaee the diaphragm until the oeeurrenee of considerable liquid effusion.

Just as pulmonary suetion diminishes as the elastie fibres of the lungs become more and more lax, so, on the other hand, does it inerease the more these fibres are stretched, for the greater this stretching, the more do the lungs strive to retract from the ehest-wall and other adjacent struetures. Hene, suetion is greater during inspiration than expiration.

The lung presses against its contiguous structures with a force equal to the pressure of the air in the air-vesicles, less the amount of elastic force these latter exert upon their contained air. The alveolar air-pressure at the end of an ordinary inspiration is about 1 millimeter Hg. less than the atmospheric pressure—*exempli gratia*, 59 millimeters Hg.—and the alveolar elastic force is then about 10 millimeters Hg. Hence, at the end of an ordinary inspiration, the lungs press upon the surrounding structures with a force equal to 749 millimeters Hg., this being 11 millimeters Hg. less than the atmospheric pressure. At the end of an ordinary expiration the alveolar air pressure is about 2 millimeters Hg. more than the atmospheric pressure—*id est*, 762 millimeters Hg.—and the alveolar elastic force about 5 millimeters Hg. Consequently, the lungs then press upon surrounding structures with a force equal to $762 - 5 = 757$ millimeters Hg., this being 3 millimeters Hg. less than the atmospheric pressure.

It is therefore clear that during ordinary breathing, the structures contiguous to the lungs are subject to suction which increases with every inspiration.

At the end of an extraordinary *in*spiration the negative pressure on the extra-pulmonary structures is considerable, while at the termination of an extraordinary *ex*piration, the pressure may become very decidedly positive. The effect of vigorous inspiration and expiration is still more pronounced when the entrance to the respiratory tract is closed.

Thus, a vigorous inspiration with completely closed mouth and nostrils may reduce the pressure to 70 millimeters Hg., or further. This is known as "Müller's experiment." The diminution of pressure is essentially due to rarefaction of the intra-pulmonary air, and not simply as in inspiration with open glottis, to stretching of the elastic lung. On the other hand, a forcible expiration with closed mouth and nostrils, after the method used by aurists to inflate the mid-ear, may raise the intra-pulmonary pressure 120 millimeters Hg. above that of the atmosphere, the heart and great blood-vessels and intra-pulmonary vessels being in this way so firmly compressed as seriously to interfere with the circulation. This is known as "Valsalva's experiment." A similar effect is produced by all forms of effort when a powerful expiration is made with partially or completely closed glottis.

The intra-pulmonary air-pressure may in a similar way be modified by causing the air which is inhaled, or that into which the expired air passes, to be condensed or rarefied.

There is, however, another factor which partially accounts for negative pressure, namely, the resiliency of the ribs or rib-spring. This was pointed out by Dr. Maguire in his paper on Dynamics of the Thorax in Disease, in the Clinical Journal of July, 1898. He instanced this resiliency by the fact that when the costal cartilages are severed in the cadaver the ribs spring outwards.

CHAPTER IX.

EMPHYSEMA.

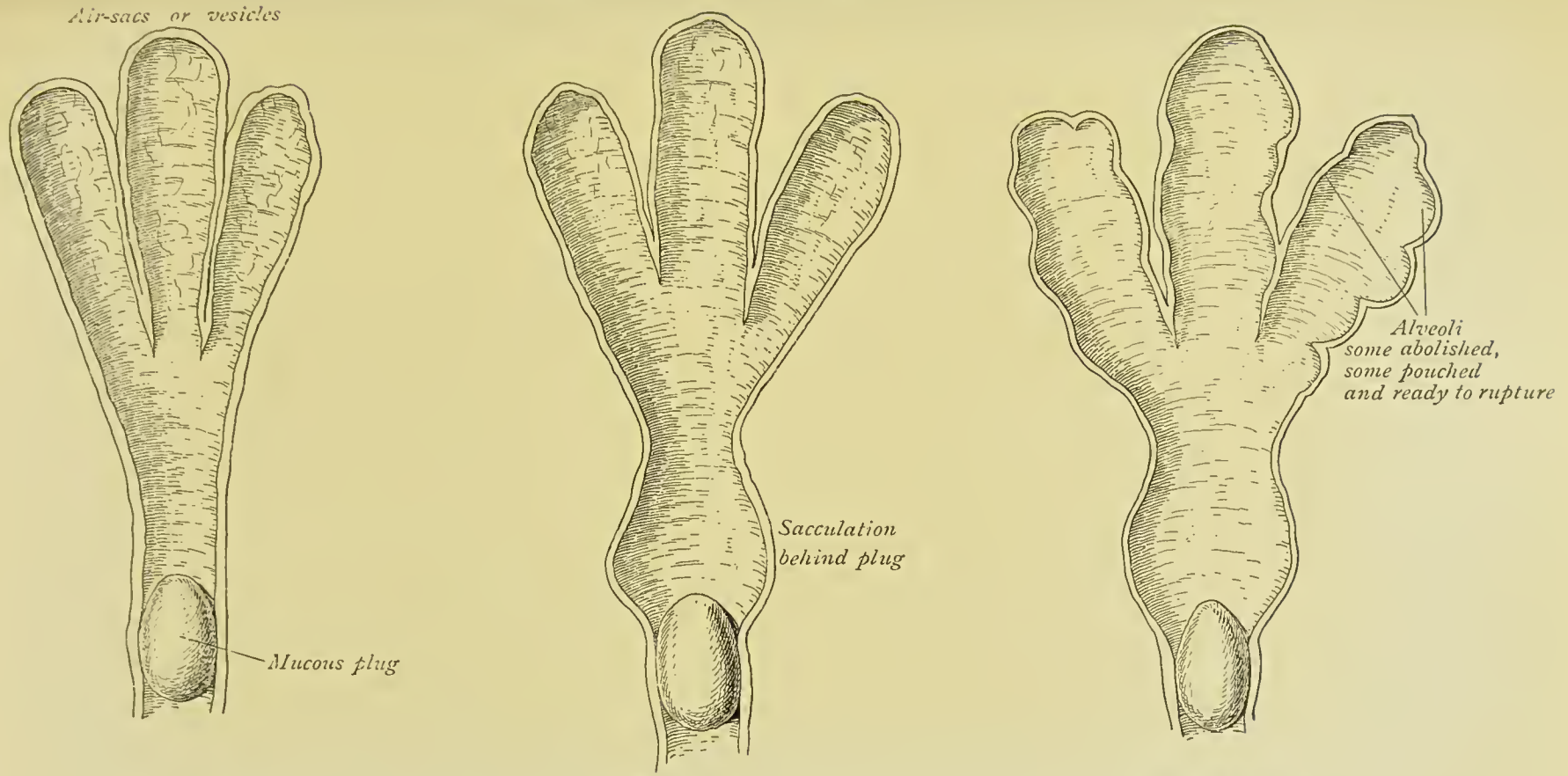
It will be well to read the chapter on "Modes in which the Thorax is Enlarged," in Campbell's book on *Respiratory Exercises*, in fact, the whole book is so teeming with practical information, that it forms a fitting prelude to the study of any thoracic derangement. Emphysema is so inseparably connected with asthma that it is necessary to consider some of its characteristics in connection with that subject. The particular variety of emphysema with which we have to do is the acute vesicular form. Osler says in his *Principles and Practice of Medicine*, "When death occurs from bronchitis of the smaller tubes, or from cyanosis when strong inspiratory efforts have been made, the lungs are large in volume and the air-cells are much distended. Clinically, this condition may develop rapidly in cases of cardiac asthma and angina pectoris. The lungs are voluminous, the area of pulmonary resonance is much increased, and on auscultation there are heard everywhere piping râles and prolonged expiration. It is the condition to which von Basch has given the names *Lungenschwellung*

and *Lungenstarrheit. A similar condition may follow pressure on the vagi.*"

Emphysema was described by Laennec and his description of the mode of its production is nearly the only item in his book on "Diseases of the Chest," which does not tally with modern thought. His theory, figured by Blake in the accompanying illustration, was "that a plug of mucus blocked up a bronchiole and prevented the air behind it from escaping; this air being retained in the body became heated so that it over-distended the alveolus. Had general physics, and more especially the Law of Expansion of Gases, been known in Laennec's day, such a view would have been impossible.

[It is generally supposed that Laennec was the first to suggest auscultation as a means of physical diagnosis, but a suggestion had already been made of immediate auscultation by the philosopher Hook of London, and published in the Posthumous Works of Robert Hook, M.D., pages 39, 40, London, 1705, folio.]

The teaching of to-day tells us that the voluntary muscles of inspiration are called more into action in dyspnœic attacks than those of expiration, owing to the fact that the appetite of the blood for oxygen is more pronounced than the necessity for getting rid of the carbon dioxide. Therefore there is a larger amount of air retained in the alveoli than normal. This air, ever increasing and



Diagrammatic Representation of Laennec's Theory of the Production of Emphysema.

accumulating, distends the alveoli, thus destroying the elasticity of the lung ; and the inspiratory muscles generally, but more especially the Scaleni, from constant contractions, become hypertrophied and shortened, and thus tend to fix the chest in a state of expansion.

Now emphysema may be caused by the dyspnœa consequent on dilatation of the heart, and on the other hand, emphysema may cause dilatation of the heart, owing to the greater difficulty with which the blood is driven through the lungs. A well-known example of emphysema, and which is due to cardiac dyspnœa, is that which occurs in Alpine guides. They take very little exercise during the winter, whilst in the summer they call upon their hearts to perform prodigies of endurance.

Emphysema is usually so connected with asthma, which indeed it can induce, that it would be futile to consider the possibility of curing the latter, unless we could discover some means of reducing the former, and no drugs have any influence over the progress of the condition itself. The lines of treatment should be as follows :

Remove any ascertainable cause, such as dilated heart, nasal obstruction, chronic constipation, hepatic congestion, indigestion, bronchitis, &c. Attend carefully to atmospheric surroundings, as emphysematous patients are very likely to contract bronchitis. But the most important treatment of any, is that by breathing exercises (see "Respiratory Exer-

cises in the Treatment of Disease," by Campbell). The object of these exercises is twofold, (1) to restore the mobility of the upper part of the thorax, and (2) to restore the lost elasticity of the lung, the absence of which acts unfavourably on the circulation owing to the absence of negative pressure; but that this elasticity can be restored is undoubted. (See Cases XII., XIII., XIV., XXV., XXX., in which decrease of emphysematous resonance and increase of thoracic expansion, have been brought about by suitable treatment.) My own special method of conducting these exercises is as follows :

The upper part of the thorax being fixed, and the breathing therefore mostly abdominal, it shall be our endeavour to modify these relations. I instruct the patient to place a towel at the back of the waist, then to quickly blow out as though he were trying to inflate a paper bag (this is to empty the lungs of their residual air), and as he does so to firmly bring the ends of the towel forward until the hands meet in such a manner that the towel shall overlap the lower ribs. The lungs are now empty and the lower ribs fixed by the constriction of the towel. I now instruct the patient, while being careful to retain the towel in its position, to inhale forcibly but slowly through the nose. As the lower part of the thorax cannot expand owing to the pressure of the towel, the upper thorax is compelled to do so, so powerful are the voluntary muscles of inspiration ; and as

the thorax is not a rigid box, but a very adaptable cage, by means of these exercises it soon recovers its normal respiratory movements, unless the costal cartilages have become fixed by ossification. I have seen quite a number of cases gain as much as two inches in chest expansion, by these means, that is to say, before the commencement of the treatment the difference between the chest measurements, in complete expiration and full inspiration at the level of the nipples, has been one inch, whereas on its completion it has increased to three inches. (See Cases XII., XIII., XIV., XXV., XXX.)

"Treatment by Deportment" must not be omitted. The emphysematous patient has high shoulders and carries his head forward and low, owing to the fact that the strong voluntary muscles of inspiration have become shortened and thickened. This condition may be greatly modified by the patient trying always to depress the shoulders, hold the head high and draw back the chin. These attitudes should always be practised, but especially during the breathing exercises before described.

There is another highly interesting, and from a diagnostic point of view, a very valuable sign in connection with emphysema, which was first figured by Blake, in a paper read before the Liverpool Congress in 1877, as the Cingula Athletica (see frontispiece), it consists in a prevascular zone along or above the insertion of the diaphragm. It

was said not to occur before middle life, and to be common amongst athletes or those who have been subjected to severe physical strain. Blake said that it was pathognomonic of gouty emphysema and was fonnd to be associated with follicular pharyngitis, with, in its later stages, congested liver and tricuspid regurgitation. It was said to bc due to pressure of the emphysematous lung, interfering with the blood-flow in the internal mammary and the intercostal veins and the Venæ azygos. This is of special interest to me, as I have observed that asthmatic patients after middle life, especially those with the gouty diathesis, invariably have this condition in a more or less marked degree (the strain in this case being supplied by the dyspnœa), and, that further, the Cingula Athletica tends to become fainter, and in some cases entirely disappears, after the reduction of the emphysema. I have frequently observed such a zone of congested vessels in the præcordium and hypochondrium in cases of cardiac dilatations, and enlargements of the liver and spleen, respectively; in fact, whercver there is interference with the blood-flow in the Venæ azygos, owing to pressure by an enlarged Viscus.

This condition was afterwards described and figured by E. Schweninger in the *Mittheilung aus der Dermatologischen Klinik, Charité Krankenhaus*, Heft 2, 1897, p. 47.

The observation of this condition would be found a very valuable corroborative aid to accurate diagnosis.

CHAPTER X.

ON THE RELATION BETWEEN CARDIAC MURMURS AND DILATATION.

After the careful observation of a great number of cases during some years of special practice, I feel justified in saying that we may put it down as an axiom, that it is *impossible for anyone to say whether any murmur be due to valvular disease or not, until the concomitant dilatation be reduced, and further, that those murmurs which I have called the "murmurs of dilatation" and which include many functional murmurs, disappear on the reduction of the dilatation, whereas those murmurs which are due to valvular disease become louder owing to the increased force of the blood-stream and to the alterations in the condition of the blood itself.* And also that the murmurs due to organic lesions of the valves are not of much consequence as long as there be no dilatation; for in cases of valvular disease, if the myocardium be unable to stand the strain, dilatation always follows. Loss of compensation being synchronous with dilatation.

It is easy to imagine how murmurs of regurgitation are brought about, as the large, dilated and flabby heart has

so little tone and consistency that the chordæ tendinæ lose their staying-power, thus the valves may be pushed hither and thither, according to the direction of the blood impulse.

But it is more difficult to imagine how murmurs apparently due to stenosis may be simulated, but that this is the case, is undoubtedly proved by the fact that these apparently obstructive murmurs sometimes disappear under treatment. They may possibly be simulated owing to the want of tone of the heart generally allowing of the edges of the valvular openings being puckered up in such a way that a fold of membrane may give rise to a murmur as the blood rushed over it, similar to that produced by stenosis (?).

I have come across three cases in my practice of apparent Aortic Disease with Mitral Symptoms, that is to say Aortic Regurgitation with its concomitant symptoms, together with a presystolic murmur best heard in the mitral area. I have not thought it worth while to record these cases, as fortunately for the patients, there has hitherto been no opportunity of examining the hearts post-mortem, but they all suffered from cardiac dilatation, the reduction of which in two of the cases, caused the presystolic murmurs to disappear, although the Aortic Regurgitation persisted, showing that the presystolic murmurs were not due to mitral stenosis. Flint first described this condition some

thirty years ago, and it has since been recognised by others, to wit, Charlewood, Turner, Guileras, Steell, Gairdner, Byrom Bramwell, Broadbent, Lees, Lespérance, Sansom, Potain and others, but it was Robert Maguire who wrote the first coherent account of the condition in a paper on "The Presystolic Murmur, Without Disease of the Mitral Valve," published in the *Medical Chronicle,* June, 1890. Flint said that the murmur was produced by the mitral flaps being unduly floated up by the blood-stream produced by the Aortic Regurgitation and thus forming an obstacle to the auriculo-ventricular stream on contraction of the auricle. Maguire combatted this in his paper referred to, and said that if this were the case then the murmur so produced should be rather the rule than the exception in Aortic Regurgitation.

He went on to say:—"In cases of considerable Aortic Regurgitation the anterior mitral cusp during diastole would be acted on by two blood streams flowing in different directions, namely, that due to the aortic regurgitation and the stream flowing through the auriculo-ventricular orifice and so made to vibrate and thus on the auricular systole, might give rise to a presystolic murmur suggestive of mitral stenosis." This sounds feasible; but I myself do not believe that the cusp would vibrate because, being fixed at its base and being pushed in one direction by the auriculo-ven-

tricular stream, its edge would meet the aortic regurgitant stream. Objects subjected to the influence of streams running in different directions tend to take up a position in the slack water between the two, and this is what possibly happens to the anterior mitral cusp in these cases. The presystolic murmur might be produced by the commencing ventriculo-auricular stream hurrying the truant edge of the cusp back into its place.

CHAPTER XI.

THE RELATIONS OF CARDIAC DILATATION TO ANGINA PECTORIS.

True angina pectoris is said to be a rare disease, and this is probably true. I have only seen one case of it in four years; but that one case is so remarkable that it is my obvious duty to record it. It was published in the *Lancet* of March 21st, 1896, in a paper on the Schott Treatment. Subjoined is Huchard's differential table between the true and spurious attacks of angina pectoris.

A comparison of this table with the features of the case quoted cannot fail to be interesting.

True Angina.	*Pseudo-angina.*
Most common between the ages of forty and fifty years.	At every age, even six years.
Most common in men. Attacks brought on by exertion.	Most common in women. Attacks spontaneous.
Attacks rarely periodical or nocturnal.	Often periodical and nocturnal.

True Angina.	*Pseudo-angina.*
Not associated with other symptoms.	Associated with nervous symptoms.
Vaso-motor form rare. Agonizing pain and sensation of compression by a vice.	Vaso-motor form common. Pain less severe; sensation of distension.
Pain of short duration. Attitude: silence, immobility.	Pain lasts one or two hours. Agitation and activity.
Lesions: sclerosis of coronary artery.	Neuralgia of nerves and cardio-plexus.
Prognosis grave, often fatal.	Never fatal.
Arterial medication.	Antineuralgic medication.

Case V.—Angina Pectoris.

A nurse, unmarried, aged 44, suffered in the autumn of 1895 from an attack of endocarditis, intensified by overwork and worry, and by a severe strain when lifting a heavy patient. There was considerable pyrexia and severe præcordial pain. During three months she sustained six attacks of angina pectoris, the first attack coming on immediately after exertion. It was preceded by high temperature and was attended by a stabbing pain in the heart, numbness of the feet and legs, and "contraction of the hands, with pain down the left arm." There was a feeling

of contraction in the chest, as though the heart were "screwed up," with a feeling of impending death, and also that it would be quite impossible to draw another breath. This lasted till 2 a.m., when the condition yielded to subcutaneous injections of ether and brandy. Another attack occurred in fourteen days, of six hours' duration, inclusive of the commencement of the aura. Four additional attacks occurred during the next two months at irregular intervals, and appeared to be brought on by indigestion. I was sent for to Nailsea, in Somersetshire, and saw her on January 11th, 1896, in consultation with the late Dr. Marshall, Senior Physician of Bristol Hospital, and her medical attendant, Mr. White, of Nailsea. The prognosis was then most grave. She was unable to move. Although not then suffering from an attack of angina pectoris, she was lying on her left side, gasping, the lips were cyanosed and there was capillary congestion in the malar regions. There was no ascertainable radial pulse, and the patient appeared to be in a moribund condition. There was a considerable amount of emphysema, but on percussing with my pleximeter, I made out a large dilatation of the heart, the left heart dulness extending beyond the mid-axillary line, and the right two inches to the right of the sternum, and the apex-beat was to be felt with the finger in a corresponding position in the sixth interspace. The first cardiac sound was hardly to be

made out and all the valves were leaking. I suggested that, as the patient had been practically given up, and I had been sent for as a last resource, and that as there was so large an amount of cardiac dilatation, a few resisted exercises should be tried. The treatment was naturally thought to be of doubtful expedience, but with the consent of the patient and her friends I proceeded with the exercises. It was a moment of high tension and reminded me of St. Paul's shipwreck on the Island of Malta, when an adder darted out of the faggots and bit the Apostle in the arm, and they thought that he would have dropped dead. I administered the exercises very carefully, first on the right arm, and then on the left, which caused a good deal of pain, as the patient had been unable to move her left arm for several days. I then gave an exercise with both arms together and allowed the patient a few minutes' rest, after which, oxygenated blood began to return to the lips and there was a distinct, though feeble, radial pulse to be felt. I then gave four more exercises, with the result that the left heart margin was brought in to the extent of one and a half inches, and the patient assumed a better colour, was able to breathe much more easily, and expressed herself as feeling better. In the light of the manifest improvement following on the exercises, the patient and her friends wished the former to undergo the

Schott treatment at my hands; but she was too ill to be moved, and it was impossible for me to remain in the West of England, so I sent down one of my trained staff, to give her resisted exercises twice daily until she should be well enough to be removed. On February the 22nd, she arrived and placed herself under my care in town, accompanied by her doctor and nurse. She underwent a six-weeks' treatment of baths and exercises, and iron and arsenic were pushed. At the beginning of March, 1896, her heart was nearly normal in size, all the regurgitant murmurs had vanished, both her pulse and general condition were very much improved, and she was able to walk and eat ordinary diet. She continued to improve, and is now matron of a small infirmary at Hampstead, and rides a bicycle with benefit and enjoyment. I saw her this morning, January the 18th, 1899, and found her heart quite normal, she tells me that she has had no attack of angina since the treatment, and that during the attacks of angina she was quite unable to move, and remained in a state of terror, waiting for death. The patient kindly tells me that if any medical man would like to ask her any questions relating to her illness she would be too pleased to answer them.

Any comment on this case would be superfluous; it should obviously be thought out on its merits and the phenomena be compared with those in Huchard's list.

I do not mean to imply that all cases of Angina pectoris are due to cardiac dilatation; but I should have signally failed in my duty to the profession, had I not recorded the above case, as at least the subject is worthy of being worked out.

We must just allude to Toxic Angina, which is due to cardiac dilatation induced by the abuse of tea, coffee and tobacco, and is characterised by vagus poisoning.

CHAPTER XII.

ASTHMA.

(INTRODUCTORY).

THE subject matter of the following chapter, was published in a booklet entitled "*On So-called Spasmodic Asthma, Considered from an Entirely New Stand-point with Regard to its Radical Cure,*" and was embodied in a paper on "The Vagus Origin of Asthma," read before the Medical Society of London, March 28th, 1898.

The theories propounded were the outcome of the observation of a number of cases, in which the asthma symptoms yielded to treatment, which was directed against dilatation of the heart.

Some critics have asserted that I deem all cases of Asthma to be due to cardiac dilatation. This is not the case, as will be clearly shown later on. But certain cases lost their asthma on reduction of the cardiac dilatation, and some theory had to be invented to account for this loss.

What I did say, however, was that all cases of asthma were due to vagal irritation; and when we consider how far-reaching the influences of the vagi are (all known causes

of asthma may be traced directly or indirectly to one or more of the many ramifications of the pneumogastric nerves), I can hardly be accused of being a man of one idea for making the above statement, upheld as it is by the French school, and by many of our own pathologists. Our campaign against asthma should be conducted on the lines of endeavouring to localise the vagal irritation, and, if possible, removing it.

This subject will be fully treated later on.

But although the symptoms of Asthma are not produced in all cases by a dilated heart, undoubtedly some of them are: (see the cases quoted in which the symptoms of asthma disappeared after the cardiac dilatations had been reduced); it is also true that every case of asthma of long standing suffers from cardiac dilatation (plus emphysema which veils the condition—see Chapter I. on Percussion), both of these conditions being probably aggravated by a long course of palliative treatment.

And so although I do not state that every case of Asthma has a dilated heart, I do say *that every case I have examined has had one; and further, that I should have been unable to diagnose the state of the heart owing to the concomitant emphysema, without the aid of my plexímeter.* The fact that cent. per cent. of my Asthma patients had suffered from cardiac dilatation may partly be accounted for by the fact that patients usually only consult me as a last resource, having

already exhausted the long list of so-called remedies, many of which materially aid asthma in producing cardiac dilatation; and it is futile to attempt to cure the asthma symptoms until the cardiac dilatation and the emphysema be done away with or, at all events, considerably reduced. (See Treatment of Emphysema).

Finally, the original cause of the asthma may have worn itself out and nothing may remain but the cardiac dilatation and emphysema to be dealt with. The following cases were specially selected from the fact that the heart dilatation seemed to be the cause of the asthma, and hence, possibly, the misconception arose, that I had stated that all cases of asthma were due to cardiac dilatation.

A good deal of indefiniteness connected with the study of asthma is due to the loose application of the term. By asthma, as considered in this work, I mean a disorder of respiration as characterised by severe paroxysms of difficult breathing, with recurrence of the attacks at more or less frequent intervals, the post-mortem appearances of the lungs showing nothing but hypertrophy of the bronchial muscles and the thickening of the mucous membrane, whereas many writers have confused cardiac and other dyspnœas with true asthma. It is the latter variety which we propose to consider.

History.

Asthma was first described by Hippocrates, who was born in the year 460 B.C. He was a physician of Cos and studied physic, in which his grandfather Nebrus was so eminently distinguished, and he improved himself by reading the tablets in the temples of the gods, where each individual had written down the diseases under which he had laboured, and the means by which he had recovered. He is said to have described asthma more than 2000 years ago pretty nearly as accurately as it is described in the text-books of to-day. (See *Berkhart* on *Asthma.*) From that day to this, the search-lights of science, though shed in many directions, have hitherto failed to dissipate the haze, which envelops the precise mechanism of that form of breathlessness which is ordinarily known as "Asthma."

The causes of asthma are manifold, whilst the phenomena are constant; these consist essentially in the spasmodic contractions of the involuntary muscles which surround the bronchioles, and of the over-action of the voluntary muscles of respiration in their efforts to overcome these contractions. Trousseau, indeed, aptly describes asthma as an "Epilepsy of the Lungs."

Asthma is, then, a symptom like headache or cough, not a separate disease. In order to emphasize the necessity of endeavouring to discover some relief from this terrible

scourge to humanity, I should like to quote a short extract from the unique monograph of Dr. Salter, sometime Physician to the Charing Cross Hospital, himself a martyr to asthma. This work was published in 1860.

"But not only is asthma not an uncommon disease, but it is one of the direst suffering. The horrors of the asthmatic paroxysm far exceed any acute bodily pain: the sense of impending suffocation, the agonizing struggle for the breath of life, are so terrible that they cannot even be witnessed without sharing in the sufferer's distress. With a face expressive of the intensest agony, unable to move, speak, or even make signs, the chest distended and fixed, the head thrown back between the shoulders, the muscles of respiration rigid and tightened like cords, the tugging and straining for every breath that is drawn, the surface pallid or livid, cold and sweating: such are the signs by which this dreadful suffering manifests itself.

"And even in the intervals of health, the asthmatic's sufferings do not cease; he goes about like his fellows and among them, but he knows he is altogether different; he bears about his disease within him wherever he goes; he knows he is struck; '*hæret lateri lethalis arundo*'; he is conscious that he is not sound—he cannot be warranted; he is not certain of a day's, perhaps not of an hour's, health; he only knows that a certain percentage of his future life must be dedicated to suffering; he cannot

make an engagement except with a proviso, and from many of the occupations of life he is cut off; the recreations, the enjoyments, the indulgences of others, he dares not take; his usefulness is crippled, his life is marred; and, if he knows anything of the nature of his complaint he knows that his sufferings may terminate in a closing scene worse only than the present.

"And not only is asthma thus comparatively common and superlatively distressing, but it is peculiarly and proverbially intractable. The asthmatic is generally looked upon as an asthmatic for life, as one who, though he should suffer many things of many physicians, would be nothing bettered but rather grow worse, and the treatment is regarded as palliative."

CHAPTER XIII.

ETIOLOGY.

The students of the immediate cause of the asthmatic paroxysm are divided into two schools: the one, fathered by Dr. Bree, believing it to be an effort on the part of the lungs to get rid of some irritating matter; the other, which has by far the most adherents, believing it to be a spasm of the bronchial muscles. I have worked out the theories contained in this book, on the latter assumption. Bree's theory was quashed by Hyde Salter in the following words:—

"Dr. Bree endeavours to show that the asthmatic paroxysm, and all the excessive muscular action that attends it, is merely an extraordinary effort to get rid of some peccant and irritating matter existing in the air-tubes, in the same way as tenesmus and spasmodic contraction of the bladder are extraordinary efforts to get rid of some source of irritation in the rectum and bladder respectively—fæces of a particularly irritating character, or a stone; that this irritating matter exists in the lungs antecedently to the attack; and that the asthmatic paroxysm is the

means and mechanism of its discharge. And this view he founds on the argument of analogy, on the fact that in continued asthma, there is some permanent and immovable source of irritation in the lungs; and, that in the great majority of cases of spasmodic asthma, there is a copious secretion of pituita towards the end of the paroxysm, with the discharge of which the attack passes off. Dr. Bree maintains his argument with a deal of ingenuity, and presses many facts into the service of his theory; but the most superficial reflection would suffice now-a-days to show that it is utterly untenable, and had Dr. Bree enjoyed the light that now shines on us from those two important points, the stethoscope and our acquaintance with excito-motory action, he would never have broached the doctrine he did. The one would have shown him the fallacy of his views, the other would have opened to him a solution of his difficulty—the stethoscope would have shown him that the conditions of an extraordinary discharging power are not present in an asthmatic attack, indeed, that the power of getting rid of anything in the lungs is very much diminished by it, and the knowledge of reflex nervous action would, in connexion with anatomy, have displayed the true nature of the disease, and made all its discrepant and scattered phenomena conspire to the production of its true and simple theory."

After very careful observation of a number of cases, I have come to the conclusion, that the sputum generated by an attack is the result of the spasm and not the cause of it; and that the "spirals" of Curschmann, the "perles" of Laennec, or the typical frothy sputum, characteristic of the cessation of an attack, are to be accounted for as follows:—On the contraction of the bronchial muscles the lumina of the bronchiæ are occluded and any mucus which may be present, becomes pressed into thin and, perhaps, twisted threads. The perles may be accounted for by irregular contraction of the muscles. During the contraction the mucous glands are unable to discharge their contents, which are pent up only to pour forth when the spasm has been relaxed. This may perhaps account for the free expectoration which usually accompanies relief from the symptoms, and the fact that the air has once more access to the bronchial tubes would account for its frothy appearance. This sputum in large cities is flecked with spicules of carbon which are absent in the country. A patient of mine, Case XXIII, told me that these very thin spirals would suddenly appear in the mouth without any cough or effort on the part of the lung to get rid of them (looking as though the ciliated epithelium had been at work), and that they *always* heralded an attack of bronchial spasm. Immediately preceding the

sense of relief, somewhat thicker spirals would appear (suggestive of gradually relaxing spasm). This would be followed by steady subsidence of the symptoms, accompanied by the coughing-up of the typical sputum, resembling beaten-up white of egg.

If these views be correct, it would be impossible for the spirals to be coughed up, as there would not be sufficient air in the lungs to allow of this, it being well known that during a severe attack the sufferer is frequently unable either to speak, cough, or even to blow his nose.

Hyde Salter says that: "by far the most common causes of asthma are whooping-cough and measles." To these we must undoubtedly now add influenza. He also quotes cases where asthma has been produced by fright and other emotions then and there, but if we refer to the table of causes of cardiac dilatation, Chapter IV., we shall find it fairly exhaustive as regards the known causes of asthma.

It is an axiom in connection with nerve-tissue, that an irritation of any one part of a nerve, may be followed by a disturbance of the function of any organ supplied by it, however remote that organ may be.

It is, however, now pretty generally conceded that the origin of asthma is to be found in the irritation of the vagal nuclei or of one or more of the many ramifications of the vagi, or of some part of the sympathetic system,

which, according to Gaskell, must be viewed as being physiologically an extension of the vagus,—(when we consider how numerous these ramifications are, we shall find that an irritation of one of them is comprised in any list that has as yet been given of the causes of asthma). As lesions of the vagi themselves are extremely rare, we must look for the cause of asthma in the parts supplied by these nerves, *exempli gratia*, the bronchial or the nasal mucous membrane, the stomach, etc., etc.

But the fact that the cause may be due to a central lesion, poisoning the vagi near their origin in the medulla oblongata, induced by the invasion of some acute disease, must not be overlooked. Dr. Mackay, in a booklet entitled "Clinical and Pathological Observations in Nervous Diseases," published in 1894, quotes a case of death from a cerebral variety of influenza on which he made an autopsy fourteen hours after death. He says :—

"The medulla was sliced transversely at intervals of five millimeters. There were found on a level with the middle and upper part of the olivary body, three foci of subpial red softening. These occurred at the site of the emergence of the pneumogastric roots on each side, and of the hypoglossal roots on the right side. The largest was that involving the roots of the right pneumogastric, which passed through the softened patch. This measured

six millimeters in its greatest length, was oval in shape, and in its centre could be made out in cross section the radicular branch of the vertebral artery associated with the vagus roots. The focus of softening on the left side was of similar shape, and measured four millimeters in its greatest diameter. The third patch of softening, occupying the groove between the olivary and pyramidal bodies, involved the root of the hypoglossal on the right side, and was about three millimeters in its greatest diameter. On making similar sections of the pons, in addition to the extreme thickening of the membranes (pia-arachnoid), there was a small blood-extravasation which stained the sheath of the fifth nerve, where this emerges from the membranes."

Now, considering that influenza is a very frequent cause indeed both of asthma and cardiac dilatation, Dr. Mackay's communication is significant. Whether it be from the origin in the medulla, from Meckel's ganglion, as in hay-fever, from a superior laryngeal, from ear-mischief through Arnold's nerve, through the pharyngeals, through the recurrent laryngeal, through pressure on the main trunk in the neck (Mr. Treves permits me to quote a case of his, in which the severe spasms of asthma were set up by "the pressure of cancerous glands in the neck, on the vagi"), through irritation of the heart, lungs, stomach,

liver, spleen, or other abdominal or pelvic viscera, or of the sympathetic system, it is difficult to evade vagal origin.

Further, there must be some comprehensive means of setting in motion even a small fraction of the muscles which envelop the billions of bronchioles described by the late Professor Rutherford as existing in the lungs. Also there is one, only one, known method of artificially inducing asthma, and that is done in the following way :—

If we chloroform a dog, and divide the left vagus and gently stimulate the proximal end with electricity, in addition to other phenomena, we produce *asthma in the right lung, and tonic contractions of the right half of the diaphragm.* Germain Sée says that the fiercest agony of dyspnœa is tetanic cramp of the diaphragm. Volkmann also found that stimulation of the vagus produced contraction of the bronchial muscles; and Osler says that some cases of emphysema are due to vagal irritation.

Hyde Salter says :—"But are the pulmonary and great thoracic vessels and the heart, the only seat of organic disease engendering asthma? By no means. Many cases are on record in which the asthma was due to organic disease of the pulmonary nervous system itself, as, for instance, a tumour or exostosis pressing upon one of the pneumogastric nerves. A case recorded by Heberden, in

which asthma was due to exostosis of the upper dorsal vertebræ, was probably of this kind. Dr. Gairdner, of Edinburgh, mentions a case characterised during life by 'frequent difficulty of respiration,' which I cannot doubt was asthmatic, and in which, after death, a neuromatous tumour of the vagus was found. It is very probable that many cases of asthma in which the cause is occult, are of this nature; but the nervous lesion is such as to elude detection or *perhaps is never sought for.*"

And again :—"Cases of peptic asthma, in which the attacks are caused by pneumogastric irritation, are so common that I think few cases could be found of true spasmodic asthma in which the disease is uninfluenced by the state of the digestive organs, while in a very large number it is entirely under their control."

Osler mentions an enlarged thymus gland as exercising a pressure on the pneumogastrics, and says at the end of a paragraph on acute vesicular emphysema (which is a variety generally associated with asthma), "A similar condition may follow pressure on the vagi."

Asthma from local causes, such as aural asthma, nasal asthma, and hay-fever, deserve to be considered separately, and a chapter will be devoted to them at the end of the book. Heredity also in some cases undoubtedly exerts an influence.

In addition to the ascertainable causes of vagal origin, however, there still remains a large class of obscure cases, whose origin has been hitherto unascertainable: I allude to that class of case which is caused by cardiac dilatation, which dilatation has not been made out owing to the emphysematous lungs which covered the heart; and I now suggest that it is precisely from these hitherto obscure cases that we shall possibly derive the light which may illuminate the obscurity. In the words of Frederick Hovenden, "It may be that we shall in part fail, but the failure will probably be a step by which others will arrive at the end we aim at."

Initial observations which led me to formulate the theories propounded in this work :—

In treating chronic heart-lesions, it happened that some few of my patients suffered from asthma: they did not consult me on account of asthma: they had already exhausted medical opinion on that score. To my intense astonishment some of these cases entirely lost their distress of breathing.

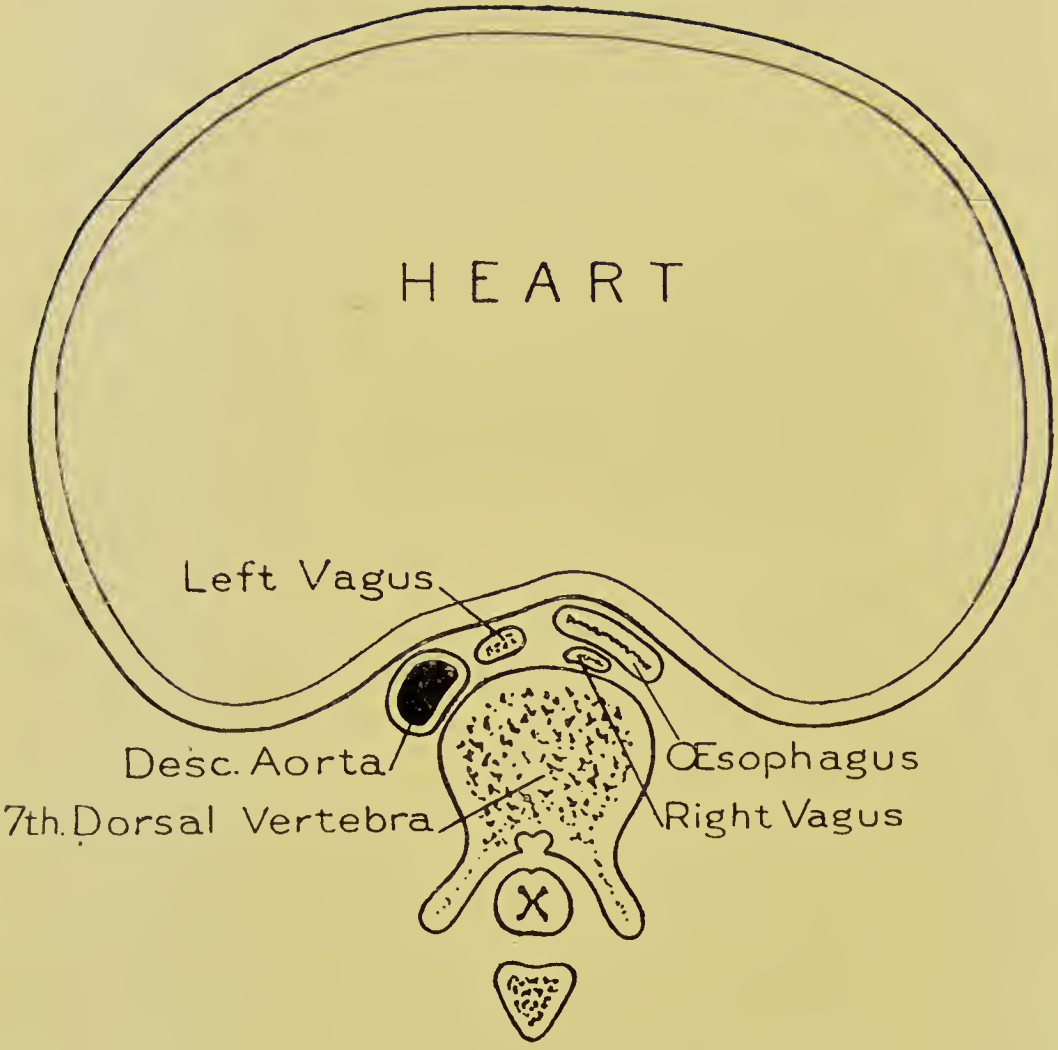
I further observed that it was precisely those cases which had no ascertainable cause of asthma that got well, and later on, I noted that those of such cases which recovered,

had invariably had a dilatation of the heart. I saw, too, that the improvement in the dyspnœa marched with the reduction of the cardiac dilatation. This set me thinking as to how the cardiac dilatation could affect these cases. I remembered the pressure-symptoms, which I had already published (see page 24, and also the experiment on the vagus of the dog, page 101) and I bethought me how the dilated heart might possibly exert, with its systole and diastole, an intermittent and, therefore, an irritating pressure on the main trunks of the pneumogastrics.

Considering that one-thirteenth of the body-weight is blood, a largely dilated heart, say to the size of a small foot-ball (not at all an uncommon condition), is a very heavy tumour; it weighs about as much as half a bucket of water, and the heart "flops" in the direction of gravity. In the supine position it can exert considerable pressure upon those structures which lie behind it, namely:—

1. The Bronchi.
2. The Œsophagus.
3. The Vagi.
4. The Descending Aorta.
5. The Vena Azygos Major.
6. The Thoracic Duct.

This pressure cannot exist until the pericardial ligaments have become stretched by the increased weight of the organ.



Diagrammatic Representation of Frozen Section of Heart at the level of the Nipples, showing the Relation of the heart to the Vagi.

On making a deep dissection of the chest, we find that the vagi, passing behind the heart, lie in close contiguity to the bony spine, so that the heavy heart can exert a pressure on their main trunks, and not only so, but the beat of the heart can hammer the nerves as though upon an anvil.

The accompanying plate is a diagrammatic representation of the relations of the heart, as altered from Braune's frozen section of the thorax, at the level of the nipples.

It is also significant that the position which nature teaches the sufferers from asthma to assume during the paroxysm, is either prone on the floor in the knee-elbow position, hanging out of the window or over a chair-back, or some similar posture, as though to relieve backward pressure. This also obtains in cases of thoracic tumour.

Now there are two obvious criticisms which naturally occur to us:

(1) If these things be, why are not such cases more frequently diagnosed?

(2) Why are not all cases of heart-dilatation complicated with dyspnœa?

To the former query I reply:

(1) In the asthmatic of any long-standing, there is

usually a large amount of emphysema, which makes accurate percussion of the heart's margin very difficult. (See Chapter I., on Percussion.)

My answer to the second question is that—

(2) If the dilatation be very great, the heart flops over on either side of the bony spine and thereby assumes a hollow conformation, somewhat resembling a saddle-back boiler, immediately over the vertebral column, by which means perhaps the vagi escape pressure.

Seeing that true asthma is due to a spasm, of the bronchial muscles, it is possible that in it we may find a solution to the problem set by the inexplicable occurrence of an attack without any apparent cause. We know that if any given muscle has frequently to perform the same kind of movement for a long time, it acquires a habit of contraction which will produce that movement. This is familiar to us in the form of "trade spasms" of various kinds, where the muscle continues to work automatically after the necessity for its action has passed away.

It is possible that such a condition may exist in the bronchial muscles of the asthmatic patient, and may perhaps produce these unexpected attacks. This is the more probable, inasmuch as in young people and in adults, who have been but recently assailed, the cause of each separate

attack is usually the more readily to be accounted for, than in cases of long-standing, suggesting that the bronchial muscles in the early cases, have not yet acquired a habit of contracting. In fact, there may be a condition of the muscle, analogous to that which is found in the subjects of cramp.

It is to be observed that in "the asthmatic" of long-standing, the lungs are seldom, if ever, *absolutely* free from the auscultatory signs of the asthmatic condition as evidenced by prolonged expiration.

Varieties of Asthma.

(Salter.)

- **Asthma**
  - Idiopathic, uncomplicated, or *Spasmodic* Asthma.
    - 1. Exciting cause manifest.
      - *Intrinsic* Asthma, *i.e.*, irritant applied to the lungs themselves.
        - I. *Asthma from fog, smoke, fumes of various kinds.*
        - II. *Ipecacuan Asthma.*
        - III. *Hay Asthma.*
        - IV. *Asthma from animal emanations.*
        - V. *Asthma from certain airs.*
        - VI. *Toxæmic Asthma.*
      - *Excito-motory*, or reflex Asthma.
        - I. *Peptic Asthma.*
        - II. *Asthma from organic nervous irritation.*
        - III. *Asthma from peripheral cerebro-spinal irritation.*
      - *Central* Asthma. Epileptic, emotional, &c.
    - 2. No apparent exciting cause of attacks. *Periodic Asthma.*
  - Symptomatic, complicated, or *Organic* Asthma.
    - Organic cause—*Vascular.*
      - I. *Asthma complicating bronchitis, common humid Asthma—senile Asthma.*
      - II. *Cardiac Asthma.*
    - Organic cause—*Nervous. Heberden's Case, &c.*

CHAPTER XIV.

TREATMENT OF ASTHMA.

Asthma never kills, that is to say, that no patient has been known to die during the paroxysm; but this would be accounted for by the fact that approaching dissolution may relax the spasm. Besides, regarding asthma as a symptom, which we do, we should no more talk of dying of asthma, than of dying of headache. My friend Dr. Goodhart holds it to be a nervous disorder; this is undoubtedly the case in many instances, the nervous disturbances being so varied and so insistent, that this theory would not be incompatible with those advanced in this work, in a large proportion of cases.

Asthmatics of long-standing are often neurotic and are frequently annoyed by being described as "hysterical." Nor need we be surprised at this. Reiterated terror and frequent want of sleep will shatter any nervous system. As Hyde Salter remarks: "The treatment has been regarded as palliative." I will give a list of the classes of remedies which are usually employed. I do not prescribe

them myself, first, because my endeavour is to try and discover the cause, and if possible, to remove it; secondly, because the asthmatic has usually been through the list on his own account and he is therefore able to determine which of the many palliatives in vogue suits his individual case, far better than any doctor could do. These palliatives include:—

1. Stimulants.
2. Depressants.
3. Sedatives.
4. Vaso-dilators.
5. Attention to diet.
6. Evacuants.
7. Mental effort.
8. Quack remedies.

It were waste of time to give in detail the long list of remedies resorted to by sufferers from asthma. I will merely mention two representatives of each class:—Stimulants, brandy and strong coffee; Depressants, tobacco pushed *ad nauseam* and tartar emetic; Sedatives, opium and stramonium; Vaso-dilators, nitrite of amyl and iodide of potassium; Evacuants, calomel and sulphate of soda; Quack remedies, it would be invidious to enumerate these, advertisements of them may be seen in the daily papers: they act mostly as local sedatives. Any one of these

remedies may relieve one sufferer, but you cannot count on its relieving the next, even if the symptoms be identical.

It is remarkable that drugs, having such contrary effects, should all in turn be of benefit to the patient; most of them merely relieve the paroxysm for a time, and many of them, as opium and alcohol, are exceedingly deleterious. The best that can be said for them as a whole is, that their effects are possibly less harmful than the continuation of the spasm.

Professor Frazer says that three-grain doses of Nitrite of Sodium have a wonderful effect on the spasm.

Dr. Wallis Ord of Salisbury tells me of two cases which obtained relief from a carefully graduated treatment by thyroid extract.

Let us see if we can throw any light on this question as to why drugs which usually have such contrary effects, should unite in relieving a definite set of symptoms. To do this, we will assume by way of hypothesis, that the views advanced in this book are correct (and this I thoroughly believe them to be), that is, that all cases of any standing, have dilatation of the heart, and that this dilatation can irritate the main trunks of the vagi and so produce asthma. (See p. 104).

Regarding the actions of the different classes of drugs from this point of view, we should say that stimulants act

by shrinking the heart (the cardio-dilating action of alcohol is an after-effect), that depressants act by lessening the force of the heart-beat, which does not irritate the nerves to the same extent, and perhaps by further dilating the heart, thus the nerves escape pressure. (See p. 106.) Sedatives act by lessening nerve-worry, if their action be general, through the centres; if local, as by inhalations, through the nerve terminations; vaso-dilators act by relieving tension; evacuants have a similar action; and as regards quack remedies, they are mostly local sedatives.

I will now proceed to define my practice in dealing with a case of asthma. The antecedents and history of the case should be thoroughly gone into, especially as regards the date of onset of the first attack and the alleged cause. The vagal centres and the regions supplied by the pneumogastrics should be carefully considered, and wherever it be possible, they should be inspected; for instance, should a patient have suffered from influenza, try and elicit any cerebral symptoms. (See p. 99.) The respiratory passages should be thoroughly examined, especially for polypi, thickened nasal mucous membrane, malformation, such as spur and deviated septum, adenoid growths and

affections of the larynx. The ears should be thoroughly examined for plugs of wax, exostoses, granulations, polypus, eczema or any other condition which might serve to irritate the vagus. Search should be made for large glands in the neck, cicatrices and cheloid, aneurysms, neuromata or any factor which might cause pressure on the nerve. The mediastinum should be carefully examined for large glands, gummatous or other tumours, and the lungs for any possible cause of irritation, also the stomach, liver, spleen, bowels, the rectum for ulcer, the anus for fissure, piles or eczema, and especially the uterus for endometritis villosa, for granular os, for polypus, or any condition which might, by retaining a purulent discharge, cause vagal poisoning by autotoxis. Other instances of retained discharge, such as the suppurating corn and antral or alveolar abscess, etc., etc., should also be sought for, and dealt with.

Last, but not least, the heart should be auscultated and carefully percussed out, and if found to be dilatated, which it probably will be, should be treated by the Schott methods.

Further, the maintenance of a cheery demeanour is of great importance in the conduct of these cases, as the question of personal influence is a very real one. All side issues should be dealt with on the ordinary lines and each symptom treated as it supervenes.

When we consider what a vast number of symptoms vagus irritation may produce, it is obvious that we must be ever on the alert to combat them. Oxygen gas inhalations should be given at least twice daily to oxygenate the blood and to relieve depression. At a later stage, the patient should be instructed in the art of breathing, in order to dilate evenly the bronchial tubes, to restore the mobility of the thorax, and to develop portions of lung which have fallen into disuse owing partly to emphysema and partly to having been pushed aside by the enlarged heart. The amount of emphysema and chest-expansion should be carefully noted and registered in order to encourage the patient by the improvement which will undoubtedly accrue. A constant war will have to be waged against the exhibition of palliatives to which the patient has probably been accustomed, and he should be instructed in obtaining relief from the paroxysm by such means as the prone position, resisted exercises, breathing exercises and inhalations of oxygen.

The inhalations of oxygen gas have a marked tendency to alleviate the paroxysm. This is conceivable, as the remote cause of asthma must be partly due to the unappeased appetite of the blood for oxygen.

Marcet, in the *Lancet* of 1895, vol. ii., p. 78, while

commenting on the beneficial influence of deep breaths in asthma, regarded a deficiency of oxygen in the blood as an important factor in its causation.

Further, if the periodicity of the asthma be marked by its coming on at a stated hour, the friends should be instructed to tide over the expected attack by distracting the mind of the patient. A fair amount of exercise should be taken, thus the patient will gain confidence by finding that he is gradually able to do more and more as others do. Soporifics should be avoided if possible; as the heart grows smaller, sleep should be encouraged by physical exercise in the open air. When the heart is reduced to its normal size and the emphysema has disappeared, the patient should be told that now that the machinery has been put in proper working order, the ultimate result will depend very much upon nature and himself. On feeling himself better, he is very apt to overdo himself, and this must be guarded against. He should be sent away after the treatment to the sea-side, preferably to Eastbourne. (See page 63.) But the best place of all for the after-cure, is that in which the patient has previously experienced immunity from attack, should such a place exist. There the factor of suggestion is likely to aid materially in the cure. He should be instructed in the use of Largiader's Exerciser, on which

he must perform at least three exercises daily, being careful to rest two minutes between each and to lie down for ten minutes afterwards. These exercises are conveniently taken before dinner.

He must also take walking exercise, at least twice a day. He should be given careful regulations regarding hill-climbing and cycling. He should be told not to be disheartened should the attacks recur, as they probably will, but that they will gradually become shorter in duration and less intense, until they ultimately disappear altogether. Further, the patient should be encouraged to write for information about any symptom which he does not understand.

There are several factors which may tend to delay recovery often for many months, sometimes for more than a year. (See Case XX.), these are :—

1. Slackness of the pericardial ligaments.
2. Want of tone of the vagi.
3. Habit-spasm of the bronchial muscles.
4. Thickening of the bronchial mucous membrane.

When the heart becomes largely dilated, it weighs considerably more than it should do, on account of the amount of blood it contains. It tends to stretch the pericardial ligaments and to allow the organ to assume positions which may seriously interfere with other structures

in the thorax. When it has been reduced to its ordinary size and weight it is possible that these stretched ligaments may take some time to regain their tone and prevent the organ from leaning against the thoracic contents.

If a nerve has been irritated for a long time it develops a state of hyperæsthesia, the date of the recovery from which, it would be impossible to predict, some patients having more recuperative powers than others. (See Case XX.).

The habit of contraction, acquired by the bronchial muscles, sometimes disappears immediately after the removal of the cause (see Case XII.) ; but this is rare, gradual recovery being the usual tendency.

A thickened mucous membrane may retard recovery for a considerable period; it is characterized by a wheezing, not as in spasmodic asthma, during expiration only, but as in bronchitis, during inspiration as well. That this thickening of the membrane disappears gradually, is probable from the breathing sounds tending to become normal in these cases after some months.

We are too apt to believe that a pathological condition once established cannot be recovered from, forgetting that the conditions during life are very different from those seen in the post-mortem room, as is evidenced by the re-

covery of the elasticity of lung-tissue after the reduction of emphysema. If the yellow elastic fibre factory has been obliged to bank its fires owing to want of demand, there is no reason that they should not be rekindled when trade becomes brisker, that is, when the conditions which obviated the exhibition of lung-elasticity have been removed.

It is well for the doctor to make up his mind that in each case he is going to obtain a favourable result. This inspires the patient with confidence, and we have all seen many a bad case pulled through by sheer determination (and the assiduity which it involves) on the part of the doctor and the attendants.

CHAPTER XV.

HAY FEVER.

Hay fever, Hay asthma or Autumnal catarrh, is thus defined by Osler: "An affection of the upper air passages often associated with asthmatic attacks, due to the action of certain stimuli upon a hyper-sensitive mucous membrane."

Hay fever is very closely allied to asthma, only with this difference, that the evidences of irritation appear in the nose and the eyes instead of being heard in the lung. It is often the precursor of asthma (see Case XXV.), and sometimes the attacks alternate with asthmatic attacks. That there is a strong neurotic element in the condition, is emphasized by the fact that J. N. Mackenzie once offered an artificial rose to smell, and so induced an attack in a woman of nervous temperament, who was subject to "rose asthma." Personally, I have seen a great number of cases, every one of them suffered from a cardiac dilatation, on the reduction of which, the tendency to hay fever invariably disappeared. (See Cases XXIV., XXV., XXVI).

REFLEX ASTHMA.

Nasal Asthma—Aural Asthma—Pharyngeal Asthma.

It is only necessary briefly to allude to these varieties, as the remarks in the foregoing chapters are applicable to them.

Briefly, should there be hypertrophy of the nasal mucous membrane, cauterize it; should there be growths or nasal spurs, or aural polypi, remove them: should there be plugs of wax in the ears, syringe them; in fact, alter any condition which tends to cause tension or obstruction.

The treatment usually recommended by the Text Books follows three lines:—

1. Nerve and heart tonics, such as strychnia, arsenic and phosphorus.

2. Change of location, every case usually having at least one place where immunity from attack can be counted on.

3. Local treatment of the nose, that is, by cauterizing and destroying the sensitive areas, but this is

very unsatisfactory. The cautery of course paralyses the nerve-end organs, but they usually regain their sensibility in a few months and the patient is as bad as ever.

In the light of my experience that every case of hay-fever that I have seen, has had a dilated heart, and that the symptoms have disappeared on the reduction of that dilatation, and that the affection cannot be entirely due to local causes, the eyes being usually affected as well as the nose, I am forced to the conclusion that we have once more to do with our old friend the vagus, whose main trunk is irritated by the pressure of the dilated heart; this, owing to the idiosyncrasy of the patient, ushers in "Hay fever," which either stands in the place of, or is followed by, bronchial asthma, according to the degree and persistence of the unfavourable cardiac conditions. I find a course of resisted exercises, extending over a fortnight, to be usually sufficient treatment to produce a favourable result in children, that is, provided there be no bronchial asthma. (See Cases XXV., XXVI., XXVII.)

I may perhaps be laying myself open to the charge, that I consider everything to be due to cardiac dilatation, but I cannot, on that account, so far pander, even to professional opinion, as to refrain from recording results

repeatedly observed and verified. The wide range of the effects of cardiac dilatation are well known, and if anyone is to be blamed it is the organ itself, and not the votaries of a system which only claims to shrink the dilated heart and thereby diminish or do away with its wide-spread effects.

CHAPTER XVI.

ILLUSTRATIVE CASES.

Cases I. to III. are examples of supposed Fatty Degeneration. Case IV. was a case of Pericardial Adhesions, which were apparently broken down by the Schott Exercises. Cases V. to XI. illustrate cardiac dilatation with murmurs, which became modified on the reduction of the dilatation.

Cases XII. to XXXIV. inclusive were cases of Asthma with Cardiac Dilatation, the Asthma disappearing on the reduction of the dilatation, whilst Cases XIII. and XIV. prove that the Asthma was due to cardiac dilatation, from the fact that on the reduction of the latter the Asthma disappeared, only to reappear when the heart was again dilated from physical shock, the Asthma again disappearing when the heart was once more shrunk. (XXV. to XXVII. were cases of Hay Asthma.) In Case XXXIII. the cardiac dilatation was due to Autotoxis, while Case XXXIV. was illustrative of the expectorated rusty sputum, without any symptoms usually associated with it.

Cases XV., XVII. to XX., and XXVIII. to XXXI.

showed the Cingula Athletica, and Cases XII. to XIV., XXV., XXX. to XXXII., and XXIV. showed increased Chest expansion after the reduction of the Emphysema. All of these cases had Emphysema more or less well marked, except Cases XXVI. and XXVII., where tender years presumably had not given time for its supervention, and Case XXXIII. where Asthma had only recently commenced.

Case of Fatty Degeneration.

Case I. Madame A., aged 50, married, consulted me on November 25th, 1897, suffering from Cardiac Failure, evidenced by very weak compressible and intermittent pulse dyspnœa on the least exertion, pain, numbness and tingling down the left arm, cold extremities, lividity of the lips, and general malaise. She has been married for thirty years and has had no children. Three years ago she had an attack of influenza which left her very weak, followed by another attack during the following year. Up to the first attack of influenza, she had enjoyed good health, with the exception of an attack of blood-poisoning brought on many years ago by a wound on the foot, from this she completely recovered. I found the heart enormously dilated, the left heart-margin being five inches outside the mid-clavicular line, the apex beat as felt by the finger corresponding in position. The heart-margin instead of being rounded was wavy and there was a well marked mitral regurgitant murmur. The diag-

nosis was that of extreme dilatation of the heart with probably fatty infiltration. I called in Sir William Broadbent, and he confirmed the diagnosis and agreed that it was a suitable case for the Schott treatment. I administered a six weeks' course, with the result that the heart dimensions became normal, the mitral bruit entirely disappeared, the first heart sound had regained its full vigour, whereas before it could scarcely be made out, all the bad symptoms disappeared together with considerable general œdema, the extremities were no longer cold and the patient developed good spirits, taking exercise freely, and tricycling at Eastbourne. I saw her yesterday, January 23rd, 1899, more than two years after the treatment, and found the heart normal, and the patient in perfect health.

Case II. In consultation with Dr. Wallis Ord of Salisbury, who very kindly supplied the following notes on the case. "A male patient, aged 59, suffering from great dyspnœa on the slightest movement, with inability to ascend steps. There was marked cyanosis of the lips, and considerable capillary congestion. He complained of feeling faint and suffocated on exertion. The pulse was intermittent and very weak; there was a very feeble first sound of the heart, but no murmur, the urine contained more than a trace of albumen. His father died suddenly of heart disease at 60. The heart was much enlarged, the dulness extending on the left nearly three inches outside

the nipple line, and the margin was wavy. The case was diagnosed as one of fatty degeneration of the walls of the heart. Treatment commenced on May 12th, 1896. Under a course of baths and exercises, the heart became normal in size, the first sound regained its vigour, the dyspnœa and the sense of suffocation disappeared, as likewise the capillary digestion, the albumen being reduced to the vanishing point in a week. After six weeks' treatment he walked to the top of Old Sarum and back, and has since been grouse shooting in the mountains of Galway." This patient has maintained the good results obtained. I have since shot with him in Ireland, and two months ago (November, 1898), he spent a week with me in London, and was perfectly well.

Case III. In consultation with Mr. Staveley, who sent me the case. A female patient, aged 33, a well-known authoress, came to me in June, 1898. She had had several attacks of influenza, and years of colossal and prolonged worry. Her heart was enormously dilated. She had cyanotic lips, general œdema, a very feeble pulse, dyspnœa and faintness on the least exertion, swollen ankles, occipital headache, miserable depression and general malaise. No albumen, no murmur, but the first heart-sound was almost inaudible. After six weeks' treatment by baths, exercises, and oxygen, all these symptoms disappeared and the patient began gradually to

regain strength. The outline of the left heart was wavy.

I saw this patient at Christmas, and although she had since the date of treatment done the work of ten women, she was quite well and the heart was normal.

These cases speak for themselves. The only special remark I have to make is to emphasize the wavy outline of the left heart in each case as percussed out with my pleximeter. I have found it so in a large number of cases of suspected fatty infiltration, and I regard it, if it be concurrent with the other known symptoms of the disease, as strong presumptive evidence of that disease.

Case of Dilatation with Pericardic Adhesions.

Case IV. Seen in April, 1896, in consultation with Dr. Wallis Ord, who kindly supplied the following notes :—

"A female patient, aged 39, married, with several children. Had had Rheumatic Fever three times. Suffered from extreme emaciation and debility, with dyspnœa and orthopnœa. The heart was found to be considerably enlarged, and there were symptoms pointing to adherent pericardium, together with retraction of the skin over the apex beat in the fifth interspace during systole. There was also a very loud systolic mitral murmur. Exercises were carefully given, with immediate reduction of the size of the heart, and of the pulse rate, with

improvement in the volume of the pulse. The third administration of exercises was followed by an attack of acute jaundice, with sudden enlargement of the liver. The exercises were stopped and the symptoms slowly subsided. On resuming the exercises a precisely similar attack occurred, and it was judged prudent to suspend the treatment in this case. The patient was unwilling to go through a course of baths.

I heard from this patient's sister last week, January 10th, 1899, that she has slowly improved without further treatment and is now quite well.

I saw this patient on the 11th of February, 1899, and found that the retraction of the skin in the fifth interspace during systole had entirely disappeared. The rupture of the adhesions probably accounted for some of the untoward symptoms which supervened during the use of the resisted exercises more than two years ago. This forms additional evidence that any treatment, which will produce such results as this, should be conducted with the greatest possible care.

Case of Aortic Disease. Disappearance of a Mitral and Persistence of Aortic Murmur.

Case V. A boy, aged 14, consulted me on February 22nd, 1897. Had consulted many other doctors, all of whom were agreed that the prognosis was most grave, and

that the boy would have to lead the life of an invalid until his death, which would probably be at a comparatively early age. When I first saw him he was suffering from cardiac dyspnœa. He had cyanosis of the lips and an earthy complexion. There was very distinct throbbing all over the thorax and neck, a pulse at the nails, and an anxious distressed look. The pulse was of the "water-hammer" character, and the second sound in the aortic area was replaced by a loud bruit. Mitral valves, too, were leaking. In addition to this there was considerable dilatation of the heart. I called in Dr. Goodhart, who confirmed the diagnosis and gave a very grave prognosis. I pointed out that I proposed to reduce the existing dilatation. He replied that he did not think it would do any good, but thought that it could do no harm. So it was determined that the boy should undergo a course of Schott treatment, which he did, together with the exhibition of oxygen gas, iron and arsenic.

At the end of the treatment (six weeks) Dr. Goodhart again saw him and wrote to me as follows:—"Eric is certainly much better, he looks healthier, has more colour, the aortic character of the pulse has disappeared and the mitral murmur is hardly to be made out. I am of opinion that he may now go to school, only it should, of course, be some small school where he could be supervised."

He was accordingly sent to school at Eastbourne, where

he continued to improve under the supervision of Dr. Harper. He was allowed to play cricket, but had someone to run for him and was only permitted to bowl a few balls. I saw him again in the autumn, when he felt and looked quite well: the heart's dimensions were normal and the mitral murmur had entirely disappeared, *but the aortic murmur was much more evident.* He remained apparently quite well until the summer of 1898, when he went home for the holidays, and his friends inadvisedly allowed him to bowl all through a cricket match. He took eight wickets for thirty-eight runs, and afterwards went in and made a number of runs himself. This long strain proved too much for his heart, which became redilated, though not to the same extent as before, as there was no recurrence of the mitral murmur, *but the aortic bruit was less marked than when he was last seen, when there was no dilatation.* I read The Riot Act, gave him half-an-hour's resisted exercises, and instructed him in the method of performing self-resisted exercises, which he was to do twice daily.

On December 8th, 1898, I had a letter from him saying: "I have been doing the exercises you set me night and morning, and feel quite well."

Case of Disappearance of Systolic Bruit.

Case VI. The notes on the following two cases were kindly supplied by Dr. Gavin Morris, of Weymouth.

"Mrs. D. came under my care at the Dispensary on October 23rd, 1894, when she complained that on getting up that morning she felt faint and had tingling in her hands and feet. She suffers much from palpitation, indigestion, and many nervous symptoms, attributed to the menopause. On examination she was found to have a throbbing impulse outside the nipple line, much epigastric throbbing and a loud systolic bruit. As her occupation involves constant interruptions and overwork, I concluded that she had hypertrophy with commencing dilatation of the left ventricle.

On December 19th, Dr. Kingscote kindly saw her at his house, where Herr Hintzman treated her on the Nauheim system. She was in a very nervous state. Pulse at commencement of exercises 110, then 100, then 92, finally 100. The most marked effect was produced on the character of the impulse and sounds; the thumping character of the impulse disappeared, the bruit over the præcordial area became almost imperceptible and the apex beat retreated to the nipple line.

December 20th. The patient looks and feels much better; the thumping sensation has entirely disappeared.

The apex beat is still half-an-inch outside the nipple and the first sound prolonged, *but no bruit is audible.* Pulse 100.

Case of Disappearance of Basic Bruit.

Case VII. "November 14, 1894. Alice S., aged 25, cook. Did not menstruate till 20 years of age. Laid up four years ago with weak heart. Never had rheumatic fever. Now complains of palpitation and shortness of breath on exertion. Menstruation irregular, generally every three weeks. Loud bruit over pulmonary area. Very 'nervous.'

"December 12. Fainted in Church. Condition not improved.

"December 19. Nauheim exercises. The pulse rate decreased from 100 (its usual rate) to 80: *the basic bruit completely disappeared.*

"Deeember 27th. The *bruit* has reappeared, but *again disappeared after a few exercises.*"

The following two cases were seen in consultation with Dr. Ord of Salisbury, and the notes on them were kindly supplied by him.

Case VIII. July 1896. "A male patient, aged 59. A spare, anæmic man, with no history of rheumatism, syphilis or influenza, came complaining of pain in the loins, general weakness, great depression of spirits, shortness of breath and a constant hacking cough. He had loss of appetite

and constipation, and there was a considerable degree of chronic pharyngeal catarrh. Usual remedies failed to relieve the symptoms. On May the 27th, his heart was found to be greatly dilated, the apex beat being in the seventh space external to the nipple line. There was a systolic murmur at apex. Urine contained no albumin. A course of Baths and Exercises reduced the heart to normal dimensions, *the systolic murmur disappeared* together with all the foregoing symptoms, and the patient is now taking active exercise and is quite well."

Case of Disappearance of Systolic Murmur.

Case IX. July 1896. "A female patient, age 30, married, one child. Five years ago began to suffer from faintness and a beating in the throat. Then began to suffer from severe pains in back, head, and through the hips. Had loss of voice after exertion, and she had flashes of light before the eyes. Hands used to swell. Was very 'short-winded,' and could not sleep at night. Appetite was bad, and she suffered from enlarged tonsils and pharyngeal catarrh. Used to feel faint after food, even when lying on the sofa all day long. In June 1895, she had a sudden attack of loss of power in left arm and leg, and her legs 'went dead' up to the knees. Her heart was extremely dilated, and there was a loud blowing systolic mitral murmur. Pulse feeble and intermittent.

"Had influenza five times, three times severely. Had an attack which was evidently Rheumatic Fever, October 6th. Commenced exercises in November, 1895, and continued up till March, 1896. She has lost all her symptoms, except that she still feels a tightness in the throat, when very tired. *The systolic murmur has quite disappeared.*"

Case of Appearance of a Presystolic Murmur.

Case X. Seen January 9th, 1899. Also in consultation. A male patient, aged 60, with considerable cardiac dilatation, was treated with Resisted Exercises only—a fortnight of which shrunk the heart to the normal point—*when a presystolic murmur developed*, seeming as though there were a mitral stenosis, which owing to the peculiar conditions consequent on the cardiac dilatation, had failed to produce a murmur until the dilatation had been reduced.

Case of Disappearance of Mitral Murmur.

Case XI. A female patient, aged 26, consulted me on the 27th of September, 1898, complaining of having completely broken down from overwork amongst the poor in the East-end of London. She had all the appearance of it; she was pale and anxious-looking, with œdema under the eyes and cyanotic lips; her complexion was earthy,

and there was a considerable amount of cardiac dyspnœa from the excitement of the interview. Her mother told me that she had suffered from an unfortunate love affair, which had caused her much and long-sustained worry. It was in order to escape from this that she overworked herself amongst the poor.

On examination I found the left heart enlarged $3\frac{1}{2}$ inches, the right border being normal; all the cardiac valves were leaking, but the loudest was a systolic murmur best heard in the mitral area are also in the back and groin. The prognosis had been very grave. She had been told, and under the then existing conditions rightly, that she could never hope to live the life of an ordinary individual, and her mother was told that she would probably die soon. When asked if anything could be done, I said that we could reduce the dilatation, but that I would refrain from giving a definite prognosis, until that had been accomplished. I gave her six weeks of Schott treatment with oxygen and iron, at the end of which time, the heart was normal in size and the pulse full and regular. The murmurs of regurgitation had disappeared, except the mitral one, which was as loud, if not louder than ever, making me say that I feared that there had been therefore mitral disease present.

The patient was then sent to Eastbourne under the care of Dr. Harper, and the general condition being so much improved and the cardiac dyspnœa absent, she was

ordered to walk twice daily on the esplanade by the sea. I went down to Eastbourne a fortnight afterwards, and saw her with Dr. Harper. A still further improvement had taken place, but the mitral murmur still persisted. She was then ordered to cycle twice daily on the flat. We saw her a week afterwards and found her still stronger, with a ruddy complexion, and we thought that the murmur was somewhat modified.

I received a letter from her mother on February 3rd, 1899, telling me that her daughter was quite well, but reminding me that I had expressed a wish to see her in three months. I found her looking the picture of health, and on examination found *that the mitral murmur had entirely disappeared.*

Case of Asthma with Emphysema.

Case XII. A clergyman, aged 36, came to me from Calcutta, in August, 1897, complaining of Asthma from infancy. I found the left heart $2\frac{1}{2}$ inches outside the mid-clavicular line and the nasal mucous membrane enormously swollen. The heart was so completely covered by emphysematous lung, that it was quite impossible to define its boundaries by the ordinary methods of percussion. This patient's asthma was now constant and he was quite unable to go out after four o'clock. I called in Sir Felix

Semon with regard to the swollen condition of the mucous membrane, who wrote as follows :—

"It is certain that his nasal mucous membrane is *enormously* swollen, and it is *possible*, that by treating it his asthma may be considerably improved or even cured. But there is always such an amount of uncertainty in these cases, that I never commit myself to any *promise*, and one must be *doubly* careful, if the asthma has been in existence a long number of years, and if such an amount of emphysema has developed as in this patient's case! I have fully explained to him the situation, and advised him to talk it over with you."

Sir Felix operated six times on him. After six weeks of baths, oxygen and exercises, the asthma had gone, and the emphysema was very much reduced, the heart being normal. The improvement in this patient's condition and the gradual cessation of the asthma *began before the series of nasal operations was commenced.* In a letter from Calcutta, dated December 23rd, the patient says :—"I am keeping perfectly well and *entirely free from asthma.* Chest expansion before treatment, 1½ ins.; after treatment, 3 ins.

Supposed cause of asthma—Whooping-cough in infancy.

Case XIII. A male patient, aged 29, consulted me in December, 1897, complaining of asthma, of twenty years' standing, which had now become constant. He had had

influenza three times, and I found the left heart two inches outside the mid-clavicular line, whilst there was a great deal of emphysema. I called in Dr. Ord, who confirmed the diagnosis and remarked on the amount of emphysema. After six weeks' treatment of baths, oxygen and exercises, the asthma disappeared, the heart became normal, and the emphysema was very much reduced, as exemplified by the fact that I again took him to Dr. Ord and asked him to examine his chest. Without any suggestion on my part, Dr. Ord exclaimed: "Why, the emphysema has nearly gone."

That same evening the patient missed a step on getting out of a hansom, and falling heavily on his face, on the pavement, reproduced the cardiac dilatation to the extent of $1\frac{1}{2}$ ins., which was immediately followed by an attack of spasmodic asthma which lasted for two days. The heart was again reduced in a week to the normal by the same treatment, and the asthma *disappeared and has not since returned*, although the patient went back to live in a house in which he had hitherto always had asthma.

I met him yesterday, February 2nd, 1899, and he told me that he had just recovered from a severe attack of pleurisy and pneumonia, *without even having had a threatening of asthma.*

Supposed cause of asthma—Heart strain from over-

rowing twenty years ago. Chest expansion before treatment, 1½ ins.; after treatment, 3 ins.

Case XIV. A male patient, aged 41, sent to me by Dr. Fletcher Little. Seen December 1st, 1897, complaining of asthma from boyhood. The left heart was dilated 3 ins., and there was much emphysema. The patient was hardly ever free from asthma. After six weeks' treatment, he became quite well, and I received a letter from his father, dated January 13th, saying:—

"My son has now been with us five days, and we have had the opportunity of observing the complete change your treatment has wrought in him, not only in general health, but *in entire loss of his asthma.*"

This patient, after having lost his asthma, had a cab accident, reproducing the heart's dilatation and asthma, both of which again disappeared simultaneously, after the treatment.

I saw this patient's sister on January 14th, 1899, who told me that her brother had since remained free from asthma.

Supposed cause of Asthma: Heart strain from overrunning when a boy.

Chest expansion before treatment, 2 in.; after, 4½ in.

Case XV. A female patient of very full habit, aged 63. Seen in consultation with Dr. Scott, of Bournemouth, in June, 1896. She had a hugely dilated heart with emphysema and suspected fatty infiltration, brought on by maternity, and accentuated by the successive deaths of three of her children. In 1887 cardiac dyspnœa supervened with great debility on the least exertion, and mitral regurgitation with periodic attacks of asthma. After six weeks' treatment, the tendency to asthma disappeared, together with the mitral murmur, and the heart became normal. On the subsequent death of a fourth child all these untoward symptoms reappeared, but the patient was unwilling to undergo a second course of treatment. There was well marked Cingula Athletica.

Supposed cause of Asthma: Repeated shocks and mental worry.

Case XVI. Also in consultation. Seen December 1st, 1896. A female patient, aged 40. Asthma came on at thirteen, which disappeared until six years ago, when it came back after four attacks of influenza. The left heart was dilated 5 ins., and the organ was supposed to be in a fatty condition, the patient being absolutely unable to withstand cold. There was much emphysema. Every autumn she had either to take to her bedroom or go to Algeria for the winter. She was incapable of any exertion.

After six weeks' treatment the heart became normal, the tendency to asthma departed and the emphysema disappeared. She was able to walk well. Subsequently she caught cold, suffered from an attack of bronchitis, *but quite unaccompanied by any asthma symptoms.*

During the following "Season" she was able to dance freely and to take a cold bath in the morning. In the autumn of the same year I received a letter from her mother, saying:—"My daughter is very ill with gastric ulcer: *still no asthma.*" I had a letter from the patient the other day telling me that she has since been quite well.

Supposed cause of asthma—Physical strain when a child.

Case of Asthma with Emphysema and Cingula Athletica.

Case XVII. A female patient, aged 71, consulted me on April the 22nd, 1897, complaining of Cardiac Dyspnœa on the least exertion, accompanied by frequent attacks of asthma, especially on assuming the supine position. The left heart was dilated four inches, and there was a mitral regurgitant murmur and well-marked Cingula Athletica and Emphysema. After six weeks' treatment the heart became normal, the asthma disappeared, together with the mitral murmur and the Emphysema. On July 6th, the improved

condition was maintained, and the patient was able to witness the Jubilee Procession, and walk home, over a mile, afterwards.

Supposed cause of asthma—Long-continued strain from nursing a dying husband, and repeated mental shocks.

Case of Asthma with Emphysema.

Case XVIII. A female patient, aged 59, consulted me on October the 29th, 1897. I found her heart largely dilated from worry and repeated attacks of influenza, with much emphysema. She suffered from asthma on the least exertion, especially in the autumn, also her cardiac dyspnœa prevented her from taking any exercise. After six weeks' treatment, the heart became normal, and she was able to walk up and down stairs with ease and without producing asthma. She wrote to me on February the 2nd, 1898, saying :—

"About the asthma: I am not troubled with it at all now, and feel much less oppression in the chest, and altogether. I can go up and down stairs and take a walk of a mile also, with pleasure." There was a well marked Cingula Athletica.

Supposed cause of Asthma—worry and repeated attacks of Influenza.

Case of Asthma with Emphysema and Cingula Athletica.

Case XIX. A female patient, aged 64, consulted me on the 28th September, 1897. I found the left heart dilated 4½ inches, with mitral regurgitation and marked dyspnœa on exertion. After five weeks' treatment, the heart became normal and the asthma disappeared, as also the mitral murmur. This patient was now able to walk about freely in London; but unfortunately whilst driving in a friend's brougham, a runaway hansom collided with it, and one of the shafts piercing the window, badly bruised the poor lady's thorax. The shock and the fright combined, caused the heart to redilate with recurrence of the asthma, cardiac dyspnœa, and mitral regurgitation. This patient had a craving to go home, and, against my advice, travelled to the country, and died a few months afterwards from heart failure. The Cingula Athletica was present, together with Emphysema.

Supposed cause of Asthma—long-continued worry and anxiety.

Case XX. A male patient, aged 56, consulted me on the 19th December, 1897, complaining of asthma of 46 years' standing. I found the left heart dilated 2 inches and on account of the emphysema present, a resonant note was obtained by means of ordinary percussion all over

the præcordium. I called in Dr. Symes-Thompson, who confirmed the diagnosis. After six weeks' treatment, the heart became normal, the emphysema had disappeared, and the asthma improved to the extent that, whereas formerly cold air produced an attack lasting some days, it now disappears on entering a warm room. On the 19th March, 1898, I heard from the Riviera that the patient was as bad as ever again. The Cingula Athletica was present.

Now, when I was writing my paper for the Medical Society, on the 28th March, 1898, a colleague told me that if I had not a failure to record I must invent one, as if nothing but success were quoted, the whole paper would read too much like an advertisement.

Keeping these suggestions in view, I wrote as follows:— "Case XX. is heaven-sent in order to save us from a subtle suggestion of selecting our cases, which inevitably occurs in connection with new departures. Case XX., however, stands quite alone in his misfortune amongst the cases quoted. He has but the sad satisfaction of posing as the exception which goes to prove the rule. However, I have not yet given up all hope of a good result even here, as it is proverbial that a long period of time is needed to recover from certain nerve lesions (*see* the various pareses), and it is just possible that the rapid relief obtained by all the other cases may have been a coincidence, and that these patients' nerves possessed extraordinary recuperative powers. Further,

there may have been some other form of irritation, such as a small and benign thoracic tumour, or there may have been some central lesion, which we had failed to make out. I have since ascertained that this patient suffered from an attack of influenza, which of course may have redilated his heart."

But on the 29th January, 1899, I met this patient's sister, who told me that her brother was steadily getting better, so that after all, this case would appear to be one of rehabilitation of nerve-function long deferred, the improvement not taking place until more than a year after the treatment.

Supposed cause of asthma—Long-continued business worries. Chest expansion before treatment, $1\frac{1}{4}$ ins.; after treatment, $3\frac{3}{4}$ ins.

Case of Asthma with Emphysema.

Case XXI. A female patient, aged 60, consulted me on 19th November, 1898, complaining of asthma of four years' standing, which came on after a severe mental shock, which left her very weak and breathless. She was free from asthma in the country, but she always had it when in London, and curiously enough, every day precisely at 4 a.m. and at 4 p.m., which is perhaps suggestive of muscle habit? (See page 106.) It is also to be noted that these hours correspond pretty accurately with the

zenith and nadir of the normal temperature. There was also much emphysema.

I found the left heart dilated 2½ inches, and there was much emphysema, the patient looked pale and cadaverous, and the radial pulse was hardly to be felt. There were no murmurs. The asthma was always relieved by the assumption of the prone position.

After six weeks' treatment the heart became normal, the complexion improved, the radial pulse became full and regular, the emphysema disappeared, also the asthma, which has not since returned. This is the more remarkable as the treatment was conducted in a house in Belgravia, where the patient had previously always suffered from asthma, indeed the treatment was begun during an attack.

Supposed cause of asthma — Mental shock. Chest expansion before treatment, 2 ins.; after treatment, 4 ins.

Case of Asthma with Emphysema followed by Peptic Asthma.

Case XXII. A female patient, aged 33, seen December 20th, 1898, complaining of asthma of twenty-two years' standing. She was in an extremely emaciated condition and had been in bed for twelve months; was very weak and unable to walk. She had had measles three times in early childhood in pretty close succession, after which she was unable to run about like other children, becoming very

tired and breathless on exertion. This was followed by inflammation of the colon, frequent "styes" and granular lids. She remembered being often very ill at night, but could not recall the exact symptoms, with the exception of "cold feet and being rubbed and given stimulants." Then came a severe attack of bronchitis and pleurisy which was followed by asthma. The patient says:—

"I can recall springing out of bed with my first attack terrified out of my wits, wondering what on earth was happening to me. At first the attacks were not very frequent and were generally worse at night, but the asthma soon gained ground and came day or night, fatigue and indigestion being the commonest causes. I was not allowed to bathe because of my heart, then I had typhoid fever. Once I was six months without asthma, and during that time I suffered from scarlet fever. Between seventeen and eighteen I was hardly out of my room for a week at a time, and when about twenty, I thought myself lucky to escape scot-free for five days at a time. Then I began to have frequent attacks of bronchitis, shiverings like ague and pleurisy, at first slightly, and then more severely. This was followed by congestion of both lungs and influenza times without number. In 1897 I was fourteen weeks in bed with it. When abroad I was as ill as at home, excepting in Switzerland, where I was rather better. Twice I had what Dr. Biden, of Hyères, said was a heart

attack pure and simple, not asthma. I have always suffered much from sore-throats of every kind, and frightful headaches. My worst asthma was generally just before the monthly periods, and was usually accompanied by very severe palpitation. Railway travelling troubled me more than anything. My father suffered from asthma for sixty years, but none of the others had it. Two boy cousins had it but grew out of it. I was free from asthma for fourteen months, and during that time was better than I had ever been while taking the American 'buffalo' cure. I had Weir-Mitchell treatment for eight weeks once, which strengthened my back and my heart, but was not effectual as regards the asthma."

I found the left heart dilated three inches, and the right heart two inches, the organ being completely covered by emphysematous lung. There were no murmurs and no albumen in the urine, but the ankles and lower eyelids were œdematous, and the pulse very feeble and intermittent. The patient was suffering from asthma at the time, and was quite unable to walk across the room. After five weeks' treatment the heart became normal, and the radial pulse full and regular, the complexion improved and the œdema disappeared from the eyelids and ankles, the asthma having vanished at the end of the third week's treatment. On the 24th of January, 1899, she was well enough to be removed to Eastbourne, where I saw her on the 5th of

February, in consultation with Dr. Harper. She had had no asthma, and the improvement was maintained in other respects. She was able to go out twice daily in a bath-chair by the sea, and felt the better for it. Her appetite had returned and she was sleeping well, also she was able to walk up and down stairs unaided. Previously to this, she had undergone a short course of massage prescribed by Dr. Harper. She is now quite free from asthma, not only by her own account, but on auscultating the chest expiration is not even prolonged. Since writing the above, I hear that this patient has had a slight attack of asthma, following a fit of severe indigestion. This, however, promptly disappeared when the latter condition was attended to.

Supposed cause of asthma—Repeated severe acute illnesses.

Case of Asthma with Emphysema followed by Hepatic Asthma.

Case XXIII. A female patient, aged 40, married, three children, consulted me on January 10th, 1898, complaining of asthma since the age of eight, getting worse at thirteen, and appeared after an attack of whooping-cough and measles, followed by an attack of rheumatic fever at thirteen. On three subsequent occasions, this patient suffered from rheumatic fever, at the ages of eighteen, twenty and twenty-four. At the age of twenty-five, the

asthma seemed to alter in type. From being persistent after exertion, it assumed an intermittent character. The patient was rather better until three years ago, when the asthma again became continuous, she was hardly free from it, especially when in London. Formerly, she never had it in London.

I found the left heart dilated $3\frac{1}{2}$ ins., and the right heart $1\frac{1}{2}$ ins., there was much general œdema, but no murmurs nor albumen, radial pulse was very feeble and the patient complained of pain down the arms and legs, and numbness. After three weeks' treatment the asthma disappeared, and after six weeks the heart became normal, the pulse regular and full, and the emphysema vanished, together with the arm and leg symptoms, the complexion from having been earthy, became fresh. The patient was delighted, and went to Eastbourne on the 25th of January. Unfortunately the thermometer was nearly at zero, and the patient got a severe chill, bringing on an acute attack of asthma.

I went down to see her, and advised her immediate return to London, as she was quite convinced that Eastbourne did not suit her. I went to see her in her house in Belgravia, and found her suffering from an acute attack of congestion of the liver, accompanied by a considerable amount of asthma. I prescribed a mustard-poultice over the liver, and 3 grains of calomel in a drachm of liquorice-powder

at bed-time. The asthma promptly disappeared, and has not since returned.

Supposed cause of asthma—Whooping-cough and measles.

The chest expansion increased from $1\frac{1}{2}$ inch before, to $3\frac{1}{2}$ inches after the treatment.

Case of Asthma.

Case XXIV. A female patient, aged 44, consulted me at the beginning of June, 1896, in consultation with Dr. Wallis Ord, of Salisbury, complaining of asthma, intense restlessness, increasing debility and excitability, shooting pains in the chest, down the left arm and in the legs, insomnia, anorexia, intense depression and biliousness. She had had influenza several times.

On examination the left heart was enlarged 2 inches, the outer margin having a wavy curve, and the first sound was very weak. There was also some hepatic enlargement and emphysema. After three weeks' treatment by baths and exercises only, the heart was normal in size, as also the liver, all the pains had disappeared, she was sleeping and eating well, and her nervous symptoms were much improved. The asthma also departed, and I now hear, nearly three years afterwards, that it has not since returned, and that the patient is quite well.

Supposed cause of asthma—Repeated attacks of influenza.

Case of Hay Fever, Asthma and Emphysema.

Case XXV. On the 8th December, 1897, I saw a male patient, aged 30, a leading light-weight jockey, in consultation with Dr. Tom Robinson, complaining of Hay fever going on to asthma, the former came on in April and lasted till October, always "starting with cold sneezing." I ascertained that the patient had had frequent attacks of conjunctivitis and sometimes eczema of the lids. He had also suffered from gonorrhœal rheumatism, indigestion and "cardiac neuralgia." He came to me in great despair, saying that the asthmatic attacks were now very liable to come on whilst riding, "interfering with his gallops," and he was afraid he should have to give up his profession, which was a very lucrative one. He had sustained several horse-accidents, had broken many bones and had been generally very much knocked about. On examination I found the left heart dilated $2\frac{1}{2}$ inches, and completely covered by emphysematous lung, the chest expansion being barely three-quarters of an inch, and the first heart-sound could with difficulty be made out; also the apex beat could not be felt with the finger, the radial pulse was feeble and intermittent. After six weeks' treatment by baths, exercises and oxygen, the heart was normal in size, the emphysema had disappeared, the chest-expansion had increased from three-quarters of

an inch to two inches, the heart sounds were normal and the pulse good, and the patient was quite free from asthma; but as he himself said, he must wait until the hot weather came (during which period he had always been worse) to know whether he was really cured or not. I heard nothing of him for more than a year, although I observed that he rode a great number of winners during the following summer. Writing last week from Newmarket, he says: "I am glad to be able to tell you that I have had no Hay fever or Asthma since your treatment."

Supposed cause of asthma—Mental excitement inseparable from the line of life, together with gonorrhœal rheumatism and repeated physical shocks. Chest expansion before treatment, 1 in.; after treatment, $3\frac{1}{2}$ ins.

Case of Hay Fever.

Case XXVI. A girl of nine, was brought to me on January 1st, 1898, complaining of hay fever, which always began in June and continued through the summer.

On examination there was nothing abnormal to be made out in the nasal passages and no cause for the hay fever except heredity. She had had hay fever from infancy. I found the left heart enlarged $1\frac{1}{2}$ ins., and thinking it unnecessary, owing to the youth of the patient, and the small amount of cardiac dilatation, to prescribe a complete course of the treatment, I ordered exercises only to be

given twice daily with the result that in a fortnight the heart became normal. The following June, when she had previously always had hay fever, there was none. Her nurse told me that while passing through a hay-field she had a threatening of it, but that she (the child's nurse) there and then performed a few resisted exercises, when the attack passed off and has not since returned.

Supposed cause of hay fever—Heredity.

Case XXVII. A boy of fourteen came to me in June, 1897, complaining of hay fever. The nurse told me that he was very apt to get it in the summer and at any time if he over-exerted himself. He was suffering from an attack when I saw him.

I found the left heart dilated 2 ins. and prescribed a course of Exercises only, as in the foregoing case, and for similar reasons. The present attack ceased after two days' exercises, and after a fortnight, the heart became normal and the hay fever has not since returned.

Supposed cause of hay fever—Overstrain while running.

Case of Asthma, Emphysema and Cingula Athletica.

Case XXVIII. A male patient, aged 47, sent to me by Dr. Symes-Thompson, consulted me in October, 1898, complaining of asthma, extreme debility, nervousness and

depression, and indigestion. The asthma began at the age of eight, after an attack of scarlet fever with dropsy. The patient seems to have suffered rather from cardiac dyspnœa than from true asthma, up to the age of eighteen, when the characteristic periodicity of the latter manifested itself. He said asthma came on nearly every night, especially if he were lying on his back, and that occasionally, but not always, it followed an attack of indigestion and was liable to be induced by a north-east wind. He had a very well marked "Cingula Athletica" (See p. 77), and I called in Dr. Blake (who was the first to draw my attention to this condition), and we took a tracing of it, hoping to get another at the conclusion of the treatment, but it had then become so faint that to take a tracing was found to be impracticable. The nervous depression was so marked in this case, and he was so liable to asthma on going into the cold air, that he was unable either to mix with his fellows or to leave the house after 4 p.m. He was very neurotic. The left heart was dilated 3 ins., and the whole organ was covered by emphysematous lung. After six weeks' treatment by Baths, Resisted Exercises, Breathing Exercises, Oxygen and Iron, the heart became normal, the asthma disappeared, as also the emphysema, and the "Cingula Athletica" became so faint that it was hardly to be made out, and the patient's general condition was very much improved in every particular. He was soon able to mix freely with his fellows

and to lunch daily at a large restaurant, and about half way through the treatment, he dined with me at my house, eat and drank well, conversed freely all the evening, and I walked home with him between ten and eleven at night, which was an exeeedingly eold one, and on visiting him the next morning, he told me he had passed a capital night, and had snffered neither from asthma nor indigestion. I was terribly afraid that this patient would suffer from relapse when he went baek to the eountry (being so neurotie that he was apt to burst into tears on the smallest provoeation), and this appeared to have been the case to a eertain extent, as I had a letter from him at the beginning of February, saying that he had had several attempts at asthma, but they had not been of sueh long duration as formerly. This ease will probably prove a parallel one to Case XX.

Supposed cause of asthma—Searlet fever.

Case of Asthma and Emphysema.

Case XXIX. A female patient, aged 41, an artist, eonsulted me in June, 1897, complaining of asthma, which seriously interfered with her work. The asthmatie attacks began in 1896, after running to eatch a train, when she experieneed extreme breathlessness, with pain in the heart. She now always has asthma on walking fast or running. I found the left heart dilated 3 ins.

with mueh emphysema (there was only a ehest expansion of 1¼ ins.) and a well-marked Cingula. After six weeks' treatment by Baths, Oxygen, Exereises and Lung Gymnasties, the tendency to asthma was gone, the heart was normal, and the general health was mueh improved, the emphysema had disappeared, the ehest expansion had arisen from 1¼ ins. to 3 ins., and the eingula had beeome very faint.

Supposed eause of asthma—Strain from running.

Case of Asthma, Emphysema and Cingula Athletica.

Case XXX. A elergyman, aged 34, eonsulted me on the 4th of July, 1898, eomplaining of asthma from infaney, whieh had now beeome eonstant, espeeially when residing in London. This patient presented the typieal high shoulders of the asthmatie with hypertrophy of the voluntary muscles of inspiration. His baek was also eurved.

On examination, I found the left heart dilated 3ins. and eompletely eovered by emphysematous lung. There was a slightly marked eingula athletiea (whieh was probably only commeneing, the patient not yet having reaehed middle age, at whieh epoeh it is said to appear). After six weeks' treatment, the heart beeame normal, the asthma

disappeared together with the emphysema, and the cingula athletica remained in statu quo.

In a letter, dated February 1st, 1899, this patient says :—"I am quite well. I have had a few threatenings of asthma, but they have come to nothing." Seen again on the 14th of February, when he told me that since writing the letter he had not even had a threatening. In a letter dated April 9th, he again assured me that he remains free from asthma.

Supposed cause of asthma—Measles, overwork and fasting. Chest expansion before treatment, 1 in.; after treatment, 3 ins.

Case XXXI. An artist, aged 68, was sent to me by Dr. Coldstream, of Florence, and consulted me on the 17th July, 1898, complaining of asthma since the age of 10, which had now become constant and was increased by exertion or cold air, spasm of the larynx having supervened during the last few years. The following are the patient's own notes of his case :—

Asthma began at 10 years of age, after bad rheumatic fever. Since then I have always been subject to it. I left Rugby in consequence of it. I have always been susceptible to cold air, especially at the seaside, which disagrees with me. When young I could acclimatize myself in three weeks to a fresh place, but I am now unable to do so. Fatigue or excitement will bring on an

attack. My remedies have been strong coffee, asthma powders, brandy in small doses, and antipyrin occasionally in severe attacks. I have sometimes slept in my clothes in a chair every night, for two months at a time. My heart was strong until I had an attack of influenza, three or four years ago."

On examination, I found his left heart dilated 4 inches and the right 1½ inches. He had much emphysema, and very well marked Cingula Athletica. There was also a considerable amount of œdema of the thorax and arms of the angio-neurotic type, alluded to on page 41, chapter V. There was also numbness and pain down the arms and legs. After three weeks' of brisk treatment, the heart became nearly normal, the emphysema was much reduced, the œdema had disappeared, the asthma had become much less frequent—there having been none at all during the last week—and the patient was able to go up and down stairs and walk about freely without inducing an attack of asthma. Unfortunately this patient had to throw up the treatment and go abroad, but I have thought that even this partial improvement was worthy of record, as there were several interesting features in the case, to wit, the patient's self-history, and the existence of the angioneurotic œdema with its subsequent disappearance.

Supposed cause of asthma—Rheumatic fever.

Case of Asthma and Emphysema.

Case XXXII. On January 8th, 1899, Dr. Helen Webb requested me to see in consultation with her, a female patient, aged 49, complaining of asthma of eight years' standing. The following are notes of the case kindly supplied by Dr. Webb, her medical adviser.

"She was a very delicate child and has never been strong. Has led a hard and anxious life with a good deal of trouble. Has always worked hard and taken much regular physical exercise. She had small-pox at the age of three and extreme debility at about puberty. At the age of thirty she fainted and for some time afterwards complained of loss of memory. In 1898 she had bad influenza, at which time the heart was feeble and afterwards dilated. In 1890 she was seen by me after a curious attack which came on while out walking. She did not remember where she was and fell down and suffered from confusion of memory for a few days, accompanied by small retinal hæmorrhages. She has always been subject to hay fever on the least exertion, but latterly she has begun to suffer from asthma and has apparently recovered completely between each attack."

On examination I found the left heart dilated $2\frac{3}{4}$ ins. and the right heart 1 in. Emphysematous lung completely covered the heart and the chest. Expansion was three-quarters of an inch. The first heart-sound was hardly to be made out, while the second was emphasized and the radial

pulse was very feeble. The complexion was earthy, and the lips cyanotic, especially on exertion. She was suffering from a typical attack of spasmodic asthma at the time, and at first she had to be carried to and from the bath, such was her state of debility. Her occupation was that of a mistress in a High School. After a week's treatment she was able to walk to and from the bath, and her asthma began to disappear. After six weeks' treatment the heart became normal, the emphysema disappeared, the chest expansion being three inches, the complexion very much improved, together with the general health. She then went to Eastbourne, where I saw her, in consultation with Dr. Webb, a week afterwards. The improvement was maintained, although on one occasion she had had an attack of asthma, for which Dr. Webb injected morphia subcutaneously. A fortnight afterwards I again saw her. She had then had no more asthma, was eating and sleeping well, was looking fresh and hearty, and had sent for her bicycle.

Supposed causes of asthma—Small-pox, influenza, anxiety.

Case XXXIII.—A female patient, aged 39, married, no children, consulted me on the 10th of May, 1898, complaining of asthma, of one year's standing, extreme debility, and susceptibility to cold.

On examination, I found a slight dilation of the heart, and angio-neurotic œdema of the thorax, a condition of

general anæmia, and an "earthy complexion," but no emphysema. For some reason or other this patient struck me as having a "poisoned" look. On making strict inquiries I elicited a history of an occasional slight and brown vaginal discharge, which naturally suggested ulceration of the cervix, and it occurred to me that this might be a case of pelvic asthma and cardiac dilatation from autotoxis, due to the absorption of a purulent discharge. This proved to be the case. I sent the patient to a Gynæcologist, who discovered a cervical ulcer, which he cured. The cardiac dilatation and the asthma then disappeared simultaneously, without any further treatment. I saw this patient six months afterwards, she looked at least 10 years younger, had a rosy complexion, and told me she felt quite a girl again.

Supposed cause of asthma—unknown.

Case of Baldness and Brain Symptoms due to Dilated Heart.

Case XXXIV. A clerk in P. & O. Company, aged 41, first seen in consultation with Dr. Blake, who published the following notes in "The Scalpel," January, 1899.

"During the past four years he has steadily lost his hair and his memory. There is a completely bald patch at the vertex six inches in diameter. Whereas he used to be ruddy there is now a marked pallor of the skin. He has deadness of

the left arm, eczema of one ankle and general slight osteo-arthritis. In 1885 he had a severe blow on his head and in the five years which succeeded he had five epileptic attacks. He now gets fits of absence but does not become unconscious. These seizures appear to start with a sense of 'looseness' in his lame left hip, then crepitation is heard in the atlo-axoid articulation, and afterwards a sense of numbness spreads from the mid-dorsal region up over the back of the head, but there is no unconsciousness. After two of these he has vomited, but he neither bit his tongue nor voided his bladder. During the same period he has had vocal illusions; that is to say evil suggestions are audibly made at his ear. These are neither delusions nor hallucinations, because they start from ordinary conversation. These symptoms were all attributed to the severe blow on the interparietal region. In 1894 he had influenza, which recurred in 1895 and again in 1897. In 1895 he had abcess in his right ear. In 1896 three small sequestra came from the right parietal bone. In 1897 he had, in addition to influenza, a very severe mental shock. It seems likely that since the first attack of 'la grippe' in 1894, he has suffered from dilated heart; this appears to have been intensified by great family trouble. When he came for advice at the end of September, the outer border of the left ventricle reached 3 ins. outside the nipple, as tested by Kingscote's Plexímeter."

He was immediately put upon the Nauheim resisted

movements. As the left ventricle receded the colour of the skin improved, the memory returned, the voices diminished, and the seizures, considered by Dr. Harry Campbell to be of the nature of "flush-storms" rather than true epilepsy, grew less severe. It was now observed that the bald scalp area was steadily shrinking, and an inch of good strong growth has since appeared.

This is the more interesting in that before the man was treated for dilated heart, many "Infallible Hair Restorers" had been employed, with no improvement in the hair whatever, whilst during the treatment of his cardiac trouble, nothing was applied to the scalp!

Query—Does this new hair bear any relation to the altered state of the heart? We know that conditions like pregnancy, which are associated with augmented ventricular vigour, lead to increase of hair-growth. On the other hand, the senile decay of the hair which clothes the upper scalp, keeps pace with degenerating arteries and diminishing ventricular contractions.

Remarks.—The preceding part of this paper was written in November. Late in December, news was received from the patient that all the symptoms were steadily diminishing in intensity. His case forms a striking example of the dangers of *post hoc et propter*. It teaches in eloquent language that cerebral symptoms are not necessarily cerebral in origin.

Case of Asthma with Rusty Sputum without Symptoms.

Case XXXV. A male patient, aged 30, was seen on the 12th May, 1897. Since the age of three, he had suffered from asthma, which began with a feverish cold. He had to leave Eton at the age of fifteen, on account of his asthma. It comes on almost every night, especially in the supine position, for this reason he often spends the night in a chair. It also comes on when he catches cold, or takes a railway journey. He was better at St. Moritz. He had had influenza and pneumonia.

On examination, I found the left heart dilated $3\frac{1}{2}$ ins., there was a great deal of emphysema, and the chest expansion was 1 in. After a fortnight's treatment all these conditions were considerably improved, and the patient had very little asthma; but I was sent for suddenly by letter, which told me that he was spitting blood. On examination, I found the expectoration resembling the rusty sputum of the clearing-up stage of croupous pneumonia. There was dulness at each lung-base and the stethoscope told of crepitatio redux. There were no rigors, no quickening of pulse rate, no temperature and absolutely no other symptoms whatsoever.

I have observed similar signs in several cases of asthma of long-standing, and I attribute it to a hypostatic

œdema of the lung bases, owing to a disturbance of the physical laws of the chest. But while this (spitting up) is going on, it is wise to take every precaution against possible septic influences, as such a case of mine, which had been attended by a colleague during my temporary absence, died from rapidly advancing acute pneumonia, with a temperature of 106°. But as a rule, I so often hear of this rusty sputum in asthmatic cases, that I now take very little notice of it.

Chest expansion before treatment, 1 in.; after treatment, 3 ins.

In conclusion, I cannot refrain from quoting from one of the Trans-Atlantic Journals, an admirable series of warnings to young practitioners on the subject of supposed "heart affection."

The paper is entitled:—

Don'ts in Connection with Heart Disease.

Don't feel called upon to give digitalis as soon as you hear a murmur over the heart. Study and treat the patient, not the murmur.

Don't conclude that every murmur means disease of the heart.

Don't forget that the pulse and general appearance of the patient often tell more than auscultation.

Don't neglect to note the character of the pulse when you feel it. Possibly you may look at the tongue to satisfy the patient; feel the pulse to instruct yourself.

Don't think that every systolic murmur at the apex indicates mitral regurgitation; every systolic murmur at the aortic interspace, aortic stenosis. The former may be trivial; the latter may be due to atheroma of the arch of the aorta.

Don't say that every sudden death was due to heart disease.

Don't forget that the most serious diseases of the heart may occasion no murmur. A bad muscle is worse than a leaky valve.

Don't examine the heart through heavy clothing.

Don't give positive opinions after one examination.—*Phila. Med. Jour.*

L'Envoi.

I do not expect my readers, should there be any, to accept these theories on the spot (though I myself fully believe in them), they present only the individual impression that has been arrived at by a careful observation of many curious, and to me, novel phenomena. But this I can say, that by a strict adherence to the principles and practice laid down in this book, I have been able to relieve a large number of sufferers from asthma, and am continuing to do so. I shall be more than satisfied if my poor efforts have the effect of inciting others to fresh lines of research in a subject which has suffered from a too long continued neglect and stagnation.

The literature of Asthma is so vast and so varied that I must refer my readers to the Index Catalogue of the Surgeon-General of the United States Army, where a complete Bibliography of the subject will be found.

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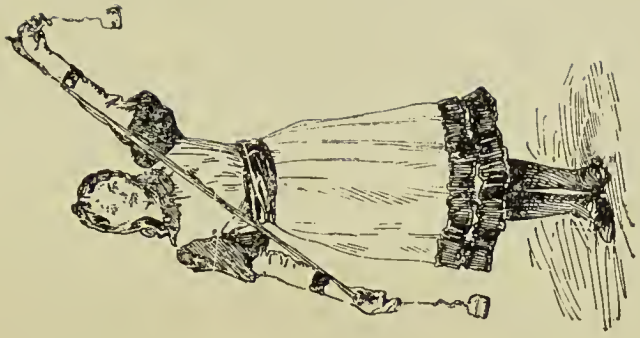

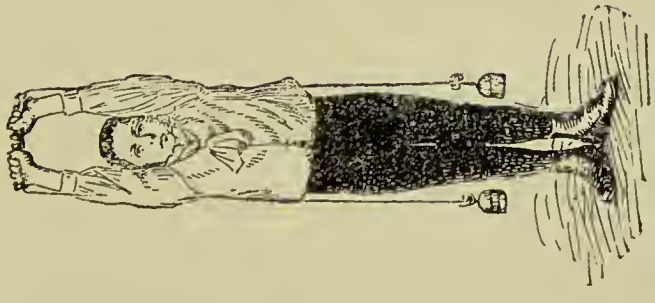

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